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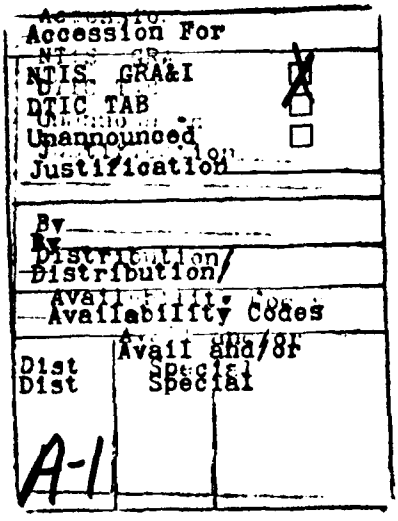

A COMPARISON BETWEEN TWO TYPES OF PREVENTIVE EDUCATIONAL PROGRAMS FOR A POPULATION AT HIGH RISK FOR CARDIOVASCULAR DISEASE

A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science
at Virginia Commonwealth University

By

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ABSTRACT

A COMPARISON BETWEEN TWO TYPES OF PREVENTIVE EDUCATIONAL PROGRAMS FOR A POPULATION AT HIGH RISK FOR CARDIOVASCULAR DISEASE

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Medical College of Virginia-Virginia Commonwealth University, 1988

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This investigation was conducted to compare the effectiveness of two types of preventive education programs in bringing about dietary changes, as measured by changes in the HDL ratios.

The questions asked were: What are the differences in the HDL ratios between participants in a Cardiovascular Risk Management (CVRM) Program and a dietary cholesterol class? What is the relationship between dietary changes reported on food frequency checklists and changes in HDL ratios?

The sample consisted of 38 males and females with elevated HDL ratios between the ages of 20 and 70+. Subjects attended either a 1-hour dietary cholesterol class (n=20) or a 5-hour CVRM program (n=18). Prior to attending the classes and 6 to 8 weeks following the intervention, all participants responded to a dietary survey that was weighted for saturated fat and cholesterol. Follow-up HDL ratios were measured prior to intervention and 6 to 8 weeks later. Each subject also completed a demographic data information sheet.

A one-way analysis of variance was used to answer the first question with a significance level set at $p = \leq .05$. No significant difference was noted between the two groups' HDL ratio means. A regression analysis was performed between the HDL ratios and dietary survey scores. The analysis indicated no correlation ($r=-0.01$, $p=.05$) between the two variables.

Dept. of Diet, Preventive Medicine, etc.

CHAPTER ONE

Introduction

Cardiovascular disease (CVD) continues to be the number one health problem in the United States. Although statistics show the incidence has dropped from 54% in 1968 (Feinleib, 1984) to 48% in 1984, CVD is still responsible for almost as many deaths as all other causes combined (AHA, 1987).

To put these statistics in perspective, before age 60, one in three U.S. males will have a cardiovascular catastrophe, i.e., a cerebrovascular accident (CVA), myocardial infarction (MI), rupture of an abdominal aortic aneurism, or similar event. MIs resulting from CVD account for over half these deaths (AHA, 1987). It is estimated that in 1987, 1.5 million Americans will have an MI and more than a third of those will die (AHA, 1987). The AHA has estimated that for 1987, it will cost $85.2 billion for medical care and lost productivity resulting just from disabling CVD.

Epidemiological advances have helped define the normal ranges for specific physiological variables within a population. At the same time, these advances have provided the basis for predicting an individual's chance of prematurely developing a given disease. For example, traditionally the normal ranges of blood pressure and serum cholesterol were determined by measuring them in healthy individuals who showed no overt signs of disease. This approach created a broad range of "normal" values. With the advent of long-term epidemiological studies, the natural history and circumstances contributing to the absence or presence of a specific disease could be observed. This has led not only to redefining the normal ranges of certain variables, but to new concepts for disease prevention (Ayers et al.,1985).

Numerous epidemiological studies have identified the variables or "risk factors" likely to be precursors to the development of CVD. The AHA has divided risk factors into three groups: (a) major risk factors that cannot be changed (heredity, sex, race, and age), (b) major risk factors

that can be changed (cigarette smoking, hypercholesterolemia, hypertension, and hyperglycemia), and (c) contributing factors, e.g., obesity, sedentary lifestyle, and stress (AHA, 1987). Note, multiple risk factors in a single individual are multiplicative rather than additive (Kannell, 1976).

Purpose of the Study

The purpose of this study is to assess whether a Cardiovascular Risk Management (CVRM) program is more effective in changing lifestyle patterns associated with CVD than other types of classes attempting to decrease the incidence of CVD.

Statement of the Problems

What are the differences in the high density lipoprotein (HDL) ratios between participants in a CVRM program and a dietary cholesterol class? What is the relationship between dietary changes reported on food frequency checklists and changes in HDL ratios?

Definition of Terms

Throughout this study the following definitions will be used:

HDL ratio - Total serum cholesterol divided by the HDL serum cholesterol. The ratio for standard risk of CVD is 5.0; the ideal ratio (approximately 3.5) corresponds to 50% of the standard risk (Kannel, 1986).

CVRM program - Two 2.5-hour informal slide presentations, covering anatomy, physiology, CVD pathophysiology and risk factors, and recommended life-style changes.

Dietary changes - Modifications of cholesterol and saturated fat intake, as measured by a questionnaire seeking information on frequency-of-consumption of specific food types.

Dietary class - A 1-hour class presentation focusing on a "prudent diet" to lower blood cholesterol levels.

Prudent diet - Nutritionally balanced diet, recommended by the American Heart Association to maintain ideal weight and includes: (a) limiting fat consumption to 30% of caloric intake with saturated fats comprising 10% and polyunsaturated not more than 10%, (b) less than 300 mg of cholesterol ingestion daily, (c) moderate salt and refined sugar intake (Haynes, 1984).

Delimitations

1. The subjects selected for this study will be from one medical facility to control for variations in CVRM programs and dietary classes and in instructors to insure consistency of content.

2. Subjects will be limited to individuals referred to the dietary and CVRM classes with elevated HDL ratios to control for other health problems which require the same type of

3. Subjects started on medication to lower cholesterol during the period of this study will be eliminated.

Assumptions

1. Behavior can be modified.
2. A pre-test/post-test method of data collection can measure changes in health behaviors.
3. Dietary changes can influence HDL ratios.
4. Laboratory enzymatic assay tests for total cholesterol and HDL cholesterol are accurate.
5. HDL ratios are predictably influenced by diet.
6. The subjects will answer the test questions truthfully.

Conceptual Framework

Webster (1986) defines adaptation as the "modification of an organism or its parts that make it more fit for existence under the conditions of its environment" (p. 55). The rise in the incidence of CVD in the last 100 years suggests that man is maladapted to his environment.

This maladaptation, or dysfunctional lifestyle, described by Haynes (1984) has been shown to result from our modern, affluent way of life, e.g., a diet high in fats and sugars, lack of physical activity, and increased stress. It is these negative health oriented behaviors which have been identified as precipitating elements (or risk factors) of cardiovascular disease (CVD). The number of risk factors within a lifestyle determine the degree to which it is dysfunctional. Unfortunately, an individual with a dysfunctional lifestyle is usually asymptomatic and therefore unaware of the developing disease.

A functional lifestyle includes activities that promote optimal cardiovascular health. It takes a conscious and continuous effort to develop healthy living habits. Progress occurs gradually and is not linear but rather one of peaks and valleys (Haynes, 1984).

A knowledge of a functional lifestyle is not enough to ensure functional living. Individuals must both perceive a need and possess a willingness to make the required changes. Haynes (1984) explains the process of needed change by using the Haynes Optimal Health Model. This model, based on the theoretical assumption that perceptions determine health habits, depicts the relationship between functional/dysfunctional lifestyles and risk factors. The dynamic process of optimal health is dependent on the individual's perceptions of "good living," risk, health, and self. To be motivated to change to a functional lifestyle, one must change one's perceptions of those four concepts, and maintenance of good health habits is dependent on the individual's continued perceptions.

With optimal health as the goal, the nurse's role then is to assist the individual in making the perceptual changes needed to adapt to a healthy or functional lifestyle. Education is necessary

for successful adaptation. The problem is to structure adult education in such a way as to meet the needs of the clientele (Agruso, 1978).

It was not until the 1960's that accumulated knowledge on adult education from related disciplines, e.g., clinical and developmental psychology, gerontology, sociology, and anthropology, began to evolve into a coherent theory of adult learning. This new theoretical model was labelled "andragogy"--the art and science of teaching adults (Knowles, 1980). Four major assumptions of andragogy differentiate it from pedagogy: (a) Adults' self concept is one of self-directedness evolving from the total dependency of an infant. (b) An adult's experiences are a rich resource for relating new learning. (c) An adult has an increasing need to learn developmental tasks that he will need in his evolving social role, i.e. nurse, parent, wife. (d) Adult learning is problem- or performance-centered, and adults learn best when teaching is oriented to particular life problems (Knowles, 1978). Thus, the nurse educator can use the theoretical framework of andragogy when selecting strategies to help individuals alter their perceptions and achieve more functional lifestyles.

Significance to Nursing

Today, CVD is the single largest cause of death in the U.S. Since nursing is the science of promoting health, the tremendous impact of CVD on our health care system is of major concern to the nursing profession. A nurse's responsibilities not only include accurately determining patients' needs and providing the appropriate care, but educating patients on how to attain and maintain their optimum state of health (Eben, 1986).

A large part of retaining or regaining one's health is determined by one's personal care habits; therefore, in order for a nurse to determine what deficit exists (so that it may be remedied), the individual's self-care practices must be considered (Kinlein, 1977) and the efficacy of alternative educational programs evaluated.

Data from this study will provide nurses with additional information relative to the most effective way of providing clients with knowledge needed to reach and maintain optimum health and to prevent or lessen the probability of developing CVD.

CHAPTER TWO

Review of Literature

By the 1970's, coronary heart disease (CHD) was responsible for one-third of all deaths in the United States. Though CHD is clearly not a new human affliction, it has become a mass phenomenon in the 20th century as lifestyles and eating habits have changed. This is, in large part, a result of meat, sugar, and dairy products replacing grains and vegetables as the primary sources of calories (Cooper, Van Horn, 1980).

According to the lipid theory of atherogenesis, developed in 1862 by Virchow, fatty substances from the blood infiltrate the arterial wall (Cooper et al. 1980). This hypothesis maintains that elevated blood lipids induce atherosclerosis and are directly responsible for atherogenesis (Feldman, 1983). Atherogenesis is the beginning of the atherosclerotic process. The process involves the thickening and hardening of the intima of the medium and large arteries due to the formation of plaques (atheromata) which are partially made up of cholesterol crystals and lipids (Langhans, 1984).

Our understanding of atherosclerosis is incomplete, but it appears that it is not an inevitable process of aging. We know that certain "risk factors" can contribute to or accelerate the onset of atherosclerosis, and it is now widely accepted that hyperlipidemia, specifically hypercholesterolemia, is a precursor to atherogenesis.

Epidemiologic Studies on CHD

One of the better known prospective epidemiological studies investigating the causes of cardiovascular disease is the Framingham Heart Study. This is a cohort study of 5,127 out of 10,000 men and women between the ages of 30 and 62 who lived in Framingham, MA in 1949. The participants have been followed biennially for the onset of CHD, stroke and other forms of

cardiovascular disease (Castelli, 1985). Periodic evaluations of prospective data have consistently shown total serum cholesterol levels to be proportionally related to increased risk of CHD (Dawber, 1980; Kannel, Castelli, & Gordon, 1970; Kannel, Castelli, Gordon, & McNamara, 1971; Kannel, Dawber, Kagan, Revotskie, & Stokes, 1961).

Other longitudinal studies on the incidence of CHD were carried out in the 1970's. The Western Collaborative Group Study followed 3,154 employed men, age 39 to 59, for the development of ischemic heart disease. Risk of CHD was studied using the multiple logistic risk model. During the 8.5 years of follow-up, 257 subjects developed ischemic heart disease. Incidence of CHD had a highly significant ($p=0.001$ for men aged 39-49; $p=0.005$ for men aged 50-59) association with serum cholesterol levels (Rosenman, Brand, Sholtz, Friedman, 1976).

The Pooling Project Research Group combined results of five longitudinal studies exploring the relationship between CHD to serum cholesterol through five steps of increasing cholesterol levels in middle-aged white men. End points of this study were fatal and nonfatal myocardial infarction, sudden cardiac death, angina, stroke, and intermittent claudication. Multivariate analysis showed total serum cholesterol, diastolic blood pressure, systolic blood pressure, and cigarette use to be independent risk factors (The Pooling Project Research Group, 1978).

The results of an epidemiological study conducted over 14.5 years by Carson and Bottiger (1981) with a sample of 3,486 Swedish men differ from many other epidemiologic studies. They found plasma triglycerides (VLDL), but not plasma cholesterol, to be an independent risk factor for the development of CHD. Clinical angina was not used as an end point for this study; the authors postulated that this may be responsible for the difference between their results and many other epidemiologic studies. Partial explanation may be that VLDL contributes to atherosclerosis, particularly in persons with other characteristics associated with an increased risk of CHD, e.g., diabetes, obesity, and low HDL (Ayers et al., 1985).

The association of CHD and hyperlipidemia has been demonstrated to occur internationally. Simons (1986) compared data from 19 of the 27 industrialized countries for which the World Health Organization had reliable mortality data from 1970 to 1980. The data came from national or regional studies of middle-aged subjects and compared CHD mortality related to interpopulation differences in lipid profiles. In men 45% of the interpopulation variation in CHD mortality was explained by variation in serum cholesterol levels; 32% by variation in HDL cholesterol; and 55% by variation in the ratio of total serum cholesterol/HDL cholesterol.

Ideal Plasma Cholesterol

The ideal plasma cholesterol level, long debated by epidemiologists, clinical investigators, and experimental pathologists, probably is in the range of 130-190 mg/dl for adults. Data from the Framingham study suggest that values of 190-200 mg/dl are associated with favorable overall health (Kannel, 1986). Review of epidemiological studies suggest a "threshold" region where the risk of CHD accelerates with rising cholesterol levels. Using the results of the Pooling Project, the American Heart Association Nutrition Committee members agreed this threshold range is approximately 200-220 mg/dl (Grundy et al., 1982).

Hyperlipidemia as a CHD Risk Indicator

The accuracy of hyperlipidemia as an indicator of CHD risk has been improved by separating the cholesterol into fractions. The current theory that low-density lipoproteins (LDL) subfractions hold the main risk of CHD and that the high-density lipoproteins (HDL) component is inversely related to risk of CHD is supported by Framingham data. Miller and Miller (1975) presented evidence that HDL cholesterol is responsible for removing plaque from the arterial walls, thus lowering the risk of CHD.

Since the LDL fraction carries most of the serum cholesterol, the serum total cholesterol is as good a CHD predictor as the LDL fraction, while being more practical to obtain. Therefore, the total cholesterol/HDL cholesterol ratio is the most accurate indicator of cholesterol transport to and from tissues, and, hence, the level of risk. A total cholesterol/HDL cholesterol ratio of 5.0 equates to the standard risk. A ratio of 3.5 corresponds to half-standard risk and is considered optimum, while a ratios between 10 and 20 indicate a 2 to 3-fold increase in risk. Research is ongoing to determine if HDL and LDL subfractions can provide more specific identification of atherogenic components (Kannel, 1986).

Numerous studies, including the Framingham and Cooperative Lipoprotein Phenotyping studies, show an inverse correlation between CHD rates and HDL levels. In high risk populations total cholesterol appears to be a stronger individual risk indicator than HDL in individuals below age 50-55. But in individuals over 55 the HDL cholesterol level was the most potent risk factor (Behari-Varga, Szekely, & Gruber, 1981; Castelli et al., 1977; Eder & Gidez, 1982; Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977; Kannel et al., 1971; Miller, G. J., & Miller, N. E., 1975; Miller, 1982; Rossner, Kjellin, Mettinger, Siden, & Soderstrom, 1978).

Rhoads, Gulbrandsen and Kagan (1976) measured serum lipoproteins in 8,006 men of Japanese ancestry living in Hawaii. Individuals with total serum cholesterol in the top quartile experienced 1.8 times the relative risk of CHD of individuals in the bottom quartile. By contrast, those with a HDL cholesterol in the bottom quartile experienced 2.2 times the relative risk of those in the upper quartile. The prophylactic effect of HDL cholesterol was independent of total serum cholesterol levels.

In a study of 82 men with angiographically documented CHD, Moberg and Wallentin (1981) demonstrated that low concentrations of HDL cholesterol were an indicator of CHD risk in normolipidemic (but not hypertriglyceridemic) males. Thirty-eight of the 82 men were normolipidemic and 22 had hypertriglyceridemia. These two groups were compared separately to

one group (n=44) of normolipidemics free of CAD and one group (n=29) of subjects with hypertriglyceridemia but no CAD. About 50% of the normolipidemic CAD individuals had HDL cholesterol in the 15th percentile of normolipidemic controls, while 65% in the same group had HDL ratios in the 15th percentile of normolipidemic controls. Almost all the hypertriglyceridemic subjects had reduced HDL cholesterol levels and elevated HDL ratios whether they had obvious CAD or not.

Diet and CHD

Various types of studies--including animal experiments, postmortum findings, dietary epidemiologic surveys, and dietary intervention trials--strongly suggest that dietary composition can influence the rate of atherogenesis and, therefore, the risk of CHD.

Animal studies (Antischkow, 1933; Katz, & Stamler, 1953; Strong & McGill, 1967; Wissler, 1974) have demonstrated that diet can cause atherogenesis by elevating plasma lipoproteins. An investigation by Vesselinovitch, Wissler, Hughs, and Borensztajn (1976) studied four groups of male rhesus monkeys that were fed high-fat, high-cholesterol diets for 18 months. Group I (n=5) was then sacrificed to assess the presence of atherogenic lesions. Group II (n=5) was fed a low-fat, low-cholesterol (reversal) diet and given an anti-atherogenic agent (W-1372) for the next 18 months. Group III (n=5) was also fed the reversal diet but without W-1372, while Group IV (n=2) was continued on the atherogenic diet. At the end of the second 18 months, the remaining animals were sacrificed and autopsies were performed on all the animals. Serum cholesterol, which had increased 5-fold with the atherogenic diet, returned to baseline values or below in Groups II and III. Autopsies also revealed 33-50% fewer lesions in the coronary arteries in Groups II and III, compared to Groups I and IV. These findings confirm previous observations (Armstrong, 1976; Armstrong, Warner, & Connor, 1970) that it is possible to achieve the regression of atherogenic lesions in primates

Other studies confirming the significant relationship between diet and atherosclerosis involve those comparing postmortum finds. Over a 20-year period, autopsies of 55-year-old Japanese males were compared with American males. Significant coronary atherosclerosis was present in 8-10% of the Japanese while 80% of the Americans had significant coronary atherosclerosis. The average Japanese diet derives 9% of its calories from fat, with 3% coming from saturated fat. By contrast, 40% of the calories in the average American diet are derived from fat, with half the calories coming from saturated fats (Stamler, 1978).

Enos et al. (1953) reported on 300 autopsies of soldiers killed in the Korean War. Most were younger than 35 and had no previously known CHD. Seventy-seven percent of the autopsied hearts displayed evidence of significant coronary artery disease. A similar investigation on 105 soldiers (mean age 22) who were killed in the Vietnam War showed evidence of CAD in 45% of the victims (McNamara, Molt, Stremple, & Cutting, 1971).

Dietary Epidemiologic Surveys

Over the past 30 years there have been numerous epidemiologic studies to determine the influence of diet on CHD. They include: Keys et al. (1958); West and Hayes (1964); McGill (1968); Keys (1970); Page, Damon and Moellering (1974); Worth, Kato, Rhoads, Kagan and Syme (1975); Shekell et al. (1981); and Kushi et al. (1985). Epidemiological surveys have advantages over intervention trials; for example, they can include large populations and duration is not a problem since subjects usually have well established eating patterns (Grundy et al., 1982).

McGill's 1968 study of the Geographic Pathology of Atherosclerosis (cited in Grundy et al., 1982) was an attempt to correlate the morbidity from atherosclerosis and dietary habits of approximately 21,000 people in 15 countries. Reviewing McGill's data, Grundy reported a high correlation between fat intake and degree of atherosclerosis.

The Seven Countries study (Keys, 1970) is the most comprehensive, prospective, international, living population study on the relationship between diet and CHD. Keys contrasted dietary composition and energy expenditure of 12,000 men from Yugoslavia, Finland, Italy, Netherlands, Greece, Japan, and the United States. Data from this study revealed a highly significant correlation (r=0.89) between saturated fat dietary intake and serum cholesterol levels for total cohorts at the entry examinations. Serum cholesterol levels and CHD rates were also significantly and positively related (r=0.81). These results are strongly suggestive that amounts of dietary saturated fats affect the incidence of CHD through their effect on plasma cholesterol.

Another project compared 475 Japanese men with similar ancestry living in Japan, Hawaii, and the United States. Diet, serum cholesterol, and frequency of CHD were the variables studied. The serum cholesterol was found to be directly related to the percentage of calories provided by fat in the diet and the incidence of CHD was directly related to the average level of serum cholesterol. Differences could not be accounted for by climate, relative obesity, physical activity, use of alcohol and tobacco, or concentration of sugar in the diet (Keys et al., 1958).

Similar findings have been duplicated in more recent cultural investigations. Page, Damon, and Moellering (1974), in a study of six Solomon Island societies, found that, though serum cholesterol did not increase with age, the highest cholesterol levels were found in the three societies that had undergone considerable Western influence. Kushi et al. studied 1,001 middle-aged-men in the 1985 Ireland-Boston Diet-Heart Study. Diet analysis showed that diets relatively high in saturated fat and cholesterol were linked to 1.6 times the relative risk of CHD, compared to diets relatively low in those substances.

Population surveys on groups of Seventh-Day Adventists, who were predominantly lacto-ovo-vegetarians, repeatedly demonstrated lower serum cholesterol levels than those seen in the general population. It appears that lower mortality rates from CHD within this group are at least partially due to dietary habits (Grundy et al., 1982).

However, not all epidemiological surveys support the association between diet serum cholesterol and incidence of CHD. For example, one study (McKeigue et al., 1985) of Asian immigrants to the United Kingdom (N=299) found no positive relationship between these factors. Although the Asians' diet had a polyunsaturated/saturated ratio of 0.85 (compared to 0.28 for the British population), they suffered a relatively high mortality from CHD. The survey was also unable to show significant differences in the two groups' total serum cholesterol and HDL cholesterol.

Nonetheless, the benchmark Western Electric study (Shekell et al., 1981) showed the correlation between diet and rates of CHD for a large population (N=1,900) of U.S. men. Dietary scores summarized intake of total cholesterol, saturated and polyunsaturated fat and found positive correlations with serum cholesterol levels and the 19-year risk of CHD. The associations withstood adjustments for confounding factors.

Large population surveys, however, have been criticized for not controlling for all other factors that might affect CHD risk. It is, therefore, necessary to consider the results of dietary intervention trials in order to determine if dietary changes can affect atherogenesis.

Dietary Intervention Trials

The first generation of clinical trials were conducted in the 1950's and 1960's. One such trial involved a group of 846 middle-aged and elderly males at the Veterans Administration domiciliary in Los Angeles. For 8 years, the subjects were placed on either a low-cholesterol, low-fat "prudent" diet (experimental group, n=424) or a "normal" American diet (control group, n=422). The groups were matched at the beginning of the study for weight, hypertension, age, tobacco use, serum lipid levels, and race. The prudent diet produced a 11% drop in serum cholesterol, while the standard diet resulted in a 5% rise. The control group had 70 fatal cardiovascular events, compared to 48 in the prudent diet group (Dayton, Pearce, Hachimoto, Dixon, & Tomiyasu,1969). This trial has been criticized for its mixed group of primary and secondary prevention, limited size,

and advanced age of its participants. Total death rates in both groups were the same (Grundy et al., 1982).

Another long-term (12-year) prevention trial involved institutionalized men and women in two mental hospitals in Helsinki, Finland. All male and female patients over 15 years of age were included in the study regardless of pre-existing CHD or other disease. The mortality from CHD and other causes was studied in this cross-over design study. In one hospital, a cholesterol-lowering diet was introduced. The other hospital served as the control and continued its use of a normal diet. After 6 years, the type of diet used in each hospital was reversed and the trial continued for 6 more years. The results linked cholesterol-lowering diet with significantly reduced mortality from CHD in men under 40 years of age but showed no difference in the two groups for older men and women (Miettinen, Turpeinen, Karvonen, Elosuo, Paavilainen, 1972).

From 1955 to 1980 more than 18 clinical trials tested cholesterol-lowering diets. The trials showed that diet alone can produce cholesterol reductions of 10-15%. Though all the studies were flawed in one way or another, they established the positive trend of decreasing CHD risk with cholesterol-lowering diets. A problem, however, remained: Too few subjects were being studied over too short a time period to draw definitive conclusions. A feasibility study carried out by the Atherosclerosis Task Force in the early 1970's concluded that a national diet/heart trial was not possible, since it would require 100,000 to 300,000 subjects and from 10 to 30 years to conduct a study with the power to definitively test the lipid hypothesis (Levy, 1986).

One way of increasing the power of studies testing the lipid hypothesis is to seek out high risk subjects, thus capturing more events. The Oslo Heart Trial (Hjermann, Velve Byre, Holme, & Leren, 1981), completed in 1981, did just that. This study involved a group of 1,232 coronary disease-free men with serum cholesterol levels greater than 290 mg/dl who were randomized into two groups: a treatment group and a routine community care group. Eighty percent of the participants were smokers. Hypertensive individuals were excluded. The intervention group

received preventive education and were placed on a lipid-lowering diet. The 5-year follow-up revealed the intervention group had a 14% reduction in cholesterol, a 60% reduction in cardiovascular events, and a significant decrease in fatal and non-fatal myocardial infarctions. Levy (1986) pointed out that the acceptance of the study was diminished by the fact that there was a 40% reduction in the number of cigarettes smoked in the intervention group. Even with multilogistic analysis suggesting that over 60% of the benefits were the result of lowering cholesterol and less than 20% from the reduction in smoking , other questions remained. It was argued that, since the study was not blind, extra attention could result in therapeutic benefits for the intervention group--the Hawthorne effect.

In the United States, a similar study attempted to measure the benefits of lowering cholesterol. The Multiple Risk Factor Intervention Trial (MRFIT) consisted of a group of 12,866 high risk men with hypertension, elevated serum cholesterol levels, and/or a history of smoking. Half the subjects were randomized to special intervention clinics for aggressive preventive treatment of their risk factors, while the other half was given routine community care. Although there were reductions in blood pressure and serum cholesterol levels in the special intervention group after 6 years, they were not statistically significant when compared to the control group (Multiple Risk Factor Intervention Trial Research Group, 1982).

However, a comparison of the MRFIT and the Oslo study (Leren, Helgeland, Hjermann, & Holme, 1983) revealed somewhat different results. Subgroup analysis of the MRFIT study disclosed a large reduction in risk of CHD in individuals who had high serum cholesterol levels and smoked but did not have hypertension. This group was not exposed to antihypertensive medication and enjoyed almost a 50% reduction in CHD. This is almost identical to the Oslo study findings where intervention was limited to smoking and lipid reduction. One conclusion was that side effects of the antihypertensive medications may have eliminated the benefits of the reduction in blood pressure.

Other multiple risk intervention trials in Europe (Levy et al., 1984) studying diet, smoking, hypertension, and physical activity also showed varying degrees of risk reduction for CHD, but, again, it is hard to attribute benefits to cholesterol reduction, especially, when other risk factors change at the same time.

Another way to test the lipid hypothesis is to use drug and diet combinations to increase the degree of cholesterol reduction. The early drug trials appeared promising in lowering cholesterol and the incidence of CHD. However, in long-term follow-up it became obvious that the side effects of cholesterol-lowering drugs created other problems (World Health Organization, Committee of Principle Investigators, 1984).

Despite all the evidence suggesting that lowering cholesterol reduces the incidence of CHD, the lack of a definitive trial has caused confusion and hesitancy among health professionals. The attempt by the Lipid Research Clinics' Coronary Primary Prevention Trial (LRC-CPPT) to control for many of the previous trials' design problems makes it a study of particular interest.

The LRC-CPPT was carefully designed as a randomized, multi-center, double-blind, placebo-controlled primary prevention trial (Lipid Research Clinics' Program, 1984). The research was carried out in 12 lipid research clinics pon a population of 3,806 over 7 years. All subjects were males between the ages of 35 and 59 and had cholesterol levels above 265 mg/dl. Those taking lipid-lowering or antihypertensive drugs were excluded, as were individuals with other diseases likely to limit life expectancy. All participants were placed on a modified low-fat, low-cholesterol diet and were seen monthly in the clinic. After five visits, they were randomized into two equal groups. One group received the cholesterol-lowering drug Cholestyramine, the other group a placebo. The treatment group had a 13% reduction in total serum cholesterol; the control group only a 1-2% reduction. The LRC-CPPT findings show that reducing total cholesterol by lowering LDL cholesterol levels diminishes incidence of CHD morbidity and mortality in men at high risk for

CHD because of raised LDL cholesterol levels. The consistency of the data of this well-designed study provides strong support for a causal role for lipids in the pathogenesis of CHD.

Another recent secondary prevention trial supports the findings of the LRC-CPPT. Conducted by the National Heart, Lung, and Blood Institute, the Type II Coronary Intervention Trial (Levy et al., 1984) used hypercholesterolemic subjects and comparative angiography to show that cholesterol reduction with diet and drug therapy is associated with a decrease in atherosclerotic plaque progression. Additionally, it showed that this change is related to both a decrease in LDL and an increase in HDL.

A similar secondary prevention study, the Leiden Intervention Trial (Arntzenius et al., 1985), used a two-year vegetarian diet for its intervention. Findings indicated that in some individuals coronary arteriosclerosis did not progress when the total/HDL cholesterol ratio was significantly lowered by dietary interventions.

What is the Ideal Diet?

Regardless of the lack of conclusive evidence suggesting a causal relationship between diet and CHD, two well-established facts remain: Dietary saturated fat and cholesterol directly raise the total serum and LDL cholesterol, and high plasma levels of both contribute directly to atherosclerosis and CHD. The wealth of data presents compelling evidence that diet affects man's risk of CHD (Grundy et al., 1982).

The American Heart Association was one of the first organizations to recommend dietary changes for the American people in an effort to lower the increasing incidence of CHD. The heart "prudent" diet, first recommended in 1961 for the general public, was a fat-modified diet which attempted to decrease the 40-50% caloric intake of fat typically seen in the American dietary pattern (Haynes, 1984). This diet reflected limiting fat consumption to 30-35% of total calories per day with less than 10% of those coming from saturated fats and a daily cholesterol intake of less

than 300 mg. The recent report of the National Cholesterol Education Program recommends that the general public decrease the intake of total fat to less than 30% of total calories and hypercholesterolemics progressively decrease their intake of total saturated fats to less than 7% of daily calories while reducing cholesterol intake to less than 200 mg/day (Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adult, 1988).

Multiple Risk Factor Intervention Programs

Although in this study the focus is specifically on diet and hyperlipidemia's causal relationship with atherogenesis, it is well established (Kannel, 1976; Pooling Project Research Group, 1978) that multiple factors can influence the progression of atherosclerosis to varying degrees. Serum lipids, obesity, glucose tolerance, smoking and hypertension were assessed in 121 patients with peripheral vascular disease. Seven had hyperlipoproteinemia, and 15 had diabetes. The same studies were performed on 31 normal controls. Peripheral vascular disease was evaluated by angiography and scored by the number and severity of lesions. It was found that the degree of atherosclerosis was related in all patients to the number of risk factors present (Vogelberg et al., 1975). A review by Malinow (1981) revealed that 11 separate studies have demonstrated regression of atherosclerotic lesions with therapeutic measures that include: diet, cessation of smoking, exercise, lipid-lowering medications, reduction of blood pressure, and long term plasma exchange.

Additionally, results from studies of cardiovascular prevention programs addressing these multiple risk factors have been positive. Brandt et al. (1977) studied 25 patients with asymptomatic peripheral vascular disease and hyperlipoproteinemia who were referred to a cardiovascular prevention program. The patients were placed on a cholesterol-lowering diet and encouraged to stop smoking, lose weight, and (if hypertensive) decrease sodium intake. Femoral

angiograms obtained at the start of the program and 13 months later confirmed that those with regression had much greater reductions in serum cholesterol levels. Reduction in blood pressure was also associated with regression of lesions.

Large-scale prevention programs have been shown to be effective in lowering risk of CHD. The Belgian Heart Disease Prevention Project (Kornitzer, DeBacker, Dramaix, & Thilly, 1980) showed that coronary risk profiles could be altered in middle-aged males through mass media health education, supplemented by face-to-face counseling in high-risk subjects. This controlled prevention trial involved 19,390 males aged 40-59 years employed by 30 Belgian industries. The industries were paired and randomized into control or intervention units. Subjects in the control units received no instruction. In the intervention factories a health education program was organized and identified high-risk subjects were given individual counseling biannually. After 2 years, the coronary risk profiles of the high-risk subjects were compared with random samples from the control and intervention groups. There was a 20% decrease in the coronary risk profiles of the high-risk intervention groups, compared to a 12.5% increase in the control groups' profiles.

In a descriptive study of a cardiovascular risk factor intervention program (very similar to the program for the CVRM group used in this study) Whitney and Boswell (1986) followed 48 patients with established CHD or whose life style reflected multiple risk factors. The participants received 5 hours of formal teaching on risk factors and life-style changes for reducing the risk of CHD. One-to-one follow-ups occurred 4-8 weeks after the classroom sessions, and intervention was then individualized. Statistical analysis showed significant improvements ($p<.001$) in serum cholesterol, serum triglycerides, fasting blood sugar, weight, and diastolic blood pressure. Additionally, though total/HDL cholesterol ratios were not measured until late in the study, the ratio fell in 12 of the 13 patients where it was measured.

Behavioral Change

It is obvious that many types of risk factor intervention programs can be successful. But what common element or elements do these programs share which makes them successful? The essential element appears to be the ability to influence long-term behavioral changes. That is, in all the above trials, risk reduction involved influencing a change in dysfunctional lifestyles by the adoption of more favorable cardiovascular health habits. Common elements of successful programs included: identifying high risk individuals, helping them identify their dysfunctional lifestyles, internalizing their process of learning to value the new behaviors (change perceptions), and providing them with the necessary skills or techniques needed to make the appropriate changes.

For the high risk individual, much of the success of CHD risk reduction depends on the voluntary adoption of more healthful behaviors. Whereas symptomatic individuals are more willing to listen, individuals free of clinical symptoms are often resistant to altering their lifestyle or practicing positive health care activities. This resistance is best overcome with education and by health care providers nurturing within their patients a sense of personal responsibility for their own cardiovascular health (Ayers et al., 1985).

In recent years, behavioral scientists and health education specialists have thoroughly investigated the issue of compliance with recommended preventive health care actions. The theoretical framework that has evolved, usually termed the Health Belief Model (HBM), is based on individual perceptions. Studies by Becker et al. (1979) support the theory that the degree to which an individual complies with recommended health actions depends on the individual's perception of susceptibility, degree of severity, benefits and barriers. Changing perceptions requires stimuli that make the individual aware of the threat. This can be done either internally, e.g., by changing perceptions of symptoms, or externally, e.g., through education by mass media campaigns.

The question for practitioners remains: What strategies best foster cooperation with recommended health actions that require permanent lifestyle changes? Just as the determinants of health behavior are mutidimensional, so are the strategies needed to accomplish this goal (Green, 1979). Becker and Maiman (1980) reviewed numerous strategies designed to enhance patient compliance. The strategies included: raising information level; "tailoring" the regimen to the individual; helping modify health-related attitudes; improving provider-patient relationships; enlisting social support; encouraging involvement by all members of the health care team; and ensuring frequent monitoring and consistency of providers.

Studies of patient education programs bear out the efficacy of these tactics. One investigation of an education program for heart patients and their families (Scalzi, Burke, & Greenland, 1980) suggested that repeated instruction improved participants' knowledge and compliance to medical regimens. Another study of long-term exercise compliance in patients who had had coronary artery bypass surgery showed that follow-up visits were essential to reinforcing knowledge and reducing barriers to compliance (Tirrell & Hart, 1980). Additionally, social support has been shown to be positively correlated to behaviors leading to healthful lifestyles (Pender & Pender, 1986; Muhlenkamp & Sayles, 1986).

Nurses as Preventive Health Care Advocates

Since health is the very essence of the nursing model, it follows that nurses should be in the vanguard of health educators promoting positive health practices. Nonetheless, there is evidence that nurses (along with other health care providers) could do a better job of educating patients. A retrospective descriptive study by Lovvorn (1980) examined "types of preventive health cues" given to high-risk individuals. Findings showed that, although practitioners (nurses and physicians) usually identified individuals to be at high-risk for CHD, they often failed to identify all existing risk factors. Consequently, the practitioners frequently gave incomplete

recommendations to the patients. Interestingly, Lovvorn's investigation showed that health consumers supported the expansion of the nurse's role in the delivery of positive health care and the management of chronic diseases.

Summary

Epidemiologic studies have confirmed hyperlipidemia to be a major factor in the premature incidence of cardiovascular disease. Further, studies have shown that serum lipid levels that previously were thought to be safe are indeed associated with a higher risk of CHD. This has led to the establishment of lower safe limits of serum cholesterol for all age groups.

The average American diet, high in saturated fat, has been shown to be associated with the high incidence of hyperlipidemia. Thus follows that decreasing the fat intake in the American diet should result in a lowering of average serum lipid levels and, in turn, a decrease in the incidence of CHD. This connection has been supported by numerous dietary intervention trials.

Primarily because of the multiplicity of etiological factors, the medical profession has been slow to promote primary prevention measures to combat premature cardiovascular disease. Intervention trials addressing the multifactoral problem of cardiovascular disease have shown promising results in lowering the risk and incidence of CHD. These programs appear to be scientifically sound, relatively easy to administer, and cost effective. More study is needed to precisely determine both the most effective intervention measures and the ultimate impact of these measures on cardiovascular morbidity and mortality.

Regardless, the gravity and magnitude of CHD argue against further delay in advocating the population-wide lifestyle changes indicated by the American Heart Association and, most recently, by the report of the National Cholesterol Education Program. Bringing about lifestyle changes requires a great deal of patient education. Although normally practitioners do not have

the time to educate patients individually, classroom instruction, followed by periodic individual follow-up visits, have proven effective.

CHAPTER THREE

Methodology

Selection of Sample

A longitudinal follow-up design was used to answer the following two questions: a) What are the differences in the HDL ratios between participants in a CVRM program and a dietary cholesterol class? b) What is the relationship between dietary changes reported on food frequency checklists and changes in HDL ratios? The setting for this study was a medium-sized Air Force hospital providing health care to a population of 47,665 active duty and retired military personnel and their dependents. The CVRM program was run by the Family Practice Out-Patient Clinic, which had 11.5 health care providers (HCP), who saw a total of 435 patients daily. The dietary cholesterol class was run by the Nutritional Medicine Department.

All subjects had elevated HDL ratios and, over a 2-month period, attended either a dietary cholesterol class or a CVRM program. The subjects in the dietary group had been referred by HCP's to a 1-hour dietary cholesterol class that was taught by a dietician. The CVRM group consisted of individuals identified as having an increased CVD risk due to high HDL ratios and had been referred by a HCP to attend two 2.5-hour sessions presented by a Family Practice physician. The distribution of this convenience sample to the diet class or CVRM program was dependent on the referring HCPs' preference and knowledge of the educational programs available in this particular health care facility.

Measurement Technique and Instrument

HDL Ratio

HDL ratios were used to identify the change in total serum cholesterol and HDL serum cholesterol levels. The Framingham data confirm that the total cholesterol/HDL cholesterol ratio is

the most accurate indicator of cholesterol transport to and from the tissues and a clear indicator of the risk of coronary heart disease. The relationship between the HDL ratio and mortality from other forms of CVD is not so definite. A total cholesterol/HDL cholesterol ratio of 5.0 equates to the standard risk of heart disease. A ratio of 3.5 corresponds to l/2-standard risk and is considered optimum, while ratios of 10-20 indicate a 2 to 3-fold increase in risk (Kannel, 1986). An IL Monarch enzymatic assay test system was used to measure the HDL ratios.

Reliability and Validity. The National Cholesterol Education Program's Laboratory Standardization Panel on Blood Cholesterol Measurement recommends ±5% as the maximum acceptable deviation from a true value (Laboratory Standardization Panel of the National Cholesterol Education Program, 1988). The College of American Pathologists Comprehensive Chemistry 1987 Survey found the accuracy of the IL Monarch enzymatic assay test system, used by the laboratory in this study, to be within 5% of the Centers for Disease Control's (CDC) reference value (Chemistry Resource Committee, College of American Pathologists, 1988).

During the 3-month data collection period, the laboratory's IL Monarch system was calibrated against a sample traceable to CDC reference material. Errors for total cholesterol and HDL cholesterol were ±5.58% and ±3.35%, respectively. For cholesterol values in the normal to moderately abnormal range (100-300mg/dl), the system's precision was 3.5% CV (coefficient of variation). Its CV for HDL was 7.5% for the low to normal range (20-60 mg/dl).

Food Frequency Checklist

A food frequency checklist was used to identify changes in eating patterns (Appendix A, p. 48). This questionnaire was designed to measure eating patterns by using a 5-point Likert-type scale to score the frequency of intake of certain food types, i.e., meat, fish, and poultry; dairy products; fats; breads and grains; vegetables; fruits; snacks; and ethnic. Participants estimated

their frequency of intake of each food item on a scale with the five choices, ranging from "rarely (less than one time a month)" to "more than once a day." They were also given the option of leaving a line blank if the item was never consumed. Cholesterol intake was estimated from the responses.

Reliability and Validity. There have been no studies done to establish reliability or validity for the Food Frequency Checklist. However, this checklist was one of several questionnaires used in the dietary portion of the federally-funded study "Cardiovascular Risk Reduction Study" conducted at the Medical College of Virginia. This checklist was developed to measure cholesterol/saturated fat intake by a registered dietician working on the study and reflects face validity by including all food groups and representing each food group appropriately.

Scoring. A registered dietician estimated the amount of cholesterol and saturated fat in each food item and assigned it to one of three groups, for low, medium, or high cholesterol/saturated fat content. For frequency of intake, the low content group had weighting factors of 1-5; the moderate group, 6-10; and the high group 11-15. The Food Frequency Checklist was scored by totalling the numbers assigned to each selected answer. The highest possible score was 415, indicating the largest cholesterol and saturated fat intake. The lowest possible score could not be predetermined due to the possibility that some items would be left blank.

Demographic Fact Sheet

A Demographic Fact Sheet was used to collect personal data on age, sex, marital status, education level, and personal and family history of heart disease.

Data Collection

The Chief of Hospital Services of the Air Force Base Hospital granted permission for data collection and obtained the Hospital Commander's approval for the study (Appendix B, p. 51). Agreement to participate in the study was obtained from each participant. The researcher explained the purpose of the study and assured confidentiality. Before agreeing to participate, each subject was given the opportunity to discuss any questions he might have with the researcher. Those patients agreeing to participate in the study signed an informed consent (Appendix C, p. 52 [Dietary Group] and Appendix D, p. 54 [CVRM Group]). Participants kept one copy of the informed consent and returned the other to the researcher.

Subjects were assigned to the diet or CVRM groups depending on their referring providers' preference and knowledge of the educational programs available in this particular health care facility. Regardless of the group in which the subject was placed, he/she was given a pretest Food Frequency Checklist and Demographic Fact Sheet to complete, and a HDL ratio was calculated from a blood sample. Diet instructions and recommendations presented in both the dietary class and the CVRM lectures were based on the AHA prudent diet.

The dietary group attended a 1-hour session in one of the hospital's classrooms. Appendix E, p. 56, is an outline of the material covered during this session. Participants completed a Food Frequency Checklist at the beginning of class, and their pre-class HDL ratios were annotated on a log. Prior to their scheduled follow-up appointment with their HCP, each participant received a second Food Frequency Checklist by mail. They returned the completed checklist to the researcher in an enclosed self-addressed stamped envelope. Results of the follow-up HDL ratio were obtained from the hospital laboratory.

The CVRM group attended two 2.5-hour sessions in one of the hospital classrooms. Appendix F, p. 57, contains the material covered in these sessions, held on the first and second Thursdays of each month. Informal didactic slide presentations were used, and the participants

received "Patient Education Manuals" containing the information presented in class. In addition to similar information on diet received by the dietary group, the CVRM group was given information on the anatomy and physiology of the cardiovascular system, the pathophysiology of CVD, risk factors of CVD, and recommended interventions. Participants completed Food Frequency Checklists before attending the first session and their pre-class HDL ratios were annotated on a log. During the scheduled follow-up appointment with the Family Practice physician, which occurred within 6 to 10 weeks, each participant completed a second Food Frequency Checklist. They returned the completed checklist to the researcher in a furnished self-addressed stamped envelope. Results of the follow-up HDL ratios were obtained from the hospital laboratory.

CHAPTER FOUR

Analysis of Data and Discussion

The purpose of this study was to assess the relative effectiveness of a specific hospital's CVRM program and diet class in changing lifestyle patterns associated with CVD and to compare dietary changes reported on food frequency checklists with changes in HDL ratios. HDL ratios and dietary questionnaires were the tools used to assess these variables.

Characteristics of the Sample

The total sample (N=38) consisted of 28 male and 10 female subjects with elevated HDL ratios, who, over a 2-month period, attended either a CVRM program (n=18) or a cholesterol diet class (n=20). All were either active duty military, retired military, or dependents of military personnel. Initially, 30 subjects in the CVRM program and 38 individuals in the diet class met the study criteria and agreed to participate. Some of the attrition of the original sample was due to subjects being reassigned, but the majority of the losses were due to the subjects not completing a follow-up food frequency survey and/or not having their follow-up blood drawn for a HDL ratio. Ages ranged from 20 to over 70, with modal age groups of 30-39 for the CVRM group and 50-59 for the diet group. Education levels ranged from "less than high school"--the smallest subgroup (n=1) for both diet and CVRM groups--to graduate degrees. Only two subjects (both in the CVRM group) were single or divorced/separated. There was a reported positive family history of heart disease in 61% (n=11) of the CVRM group and in 45% (n=9) of the diet group. A positive personal history of heart disease was reported in 16% (n=3) of the CVRM group and 15% (n=3) of the diet group (Table 1).

Table 1

Summary of Sample (N=38) Characteristics

	CVRM Group (n=18)	Diet Group (n=20)
Sex		
Male	15	13
Female	3	7
Age		
20-29	1	1
30-39	7	2
40-49	5	7
50-59	3	8
60-69	2	1
70+	1	-
Education level		
Less than high school	1	1
High school	3	4
Some college	3	5
Associate degree	4	4
Four-year degree	3	4
Graduate degree	4	2
Marital status		
Single	1	-
Married	16	20
Divorced/separated	1	-
Family history of heart disease		
Yes	11	9
No	7	11
History of heart disease		
Yes	3	3
No	15	17

Eighteen subjects in the CVRM program and 20 subjects in the diet classes completed all the criteria for the study. Six of the 18 subjects in the January diet class also attended the February CVRM classes prior to participating in the follow-up data collection (a food frequency checklist and a blood sample to determine HDL ratio). This subgroup, termed the diet/CVRM group, was analyzed separately and in combination with the diet group. At the time of their second data collection, this group had neither the advantage of the individual follow-up CVRM program visit nor sufficient time for their HDL ratios to reflect the dietary changes taught in the CVRM classes; therefore, one could theorize that the changes in HDL ratios may have been attributable to the

initial diet group instruction. (And, in fact, the Wilcoxon's Signed Ranks Test [Table 3] indicated no statistically valid difference between the HDL ratio means of either of these diet subgroups.)

Differences between baseline and follow-up ratios ranged from -1.5 to 2.5. The difference between the sample's (N=38) baseline HDL ratio mean and follow-up HDL ratio mean was 0.65, with a standard deviation of 1.25 (Table 2).

Table 2

Summary of HDL Ratio Data (N=38)

Variables	Baseline Mean	Follow-up Mean	Mean of the Difference	S.D. of Difference
Total (N=38)	6.27	5.60	0.65	1.25
CVRM (n=18)	6.69	5.83	0.86	1.35
Diet (n=20)	5.88	5.37	0.45	1.15
Diet (n=14)	5.72	5.12	0.53	1.15
Diet/CVRM (n=6)	6.25	5.97	0.28	1.25

A Wilcoxon's signed ranks test (Table 3) was applied to answer the question: Is there a significant difference between the baseline and follow-up HDL ratio means within groups?

Table 3

Summary of Wilcoxon's Signed Ranks Test (N=38)

Variables	No. of Pairs (n)	Smaller Rank Total (R)	Critical value for P=.05
CVRM (n=18)	18	34.5	40.0
Diet (n=20)	20	63.0	60.0
Diet only (n=14)	14	28.0	21.0
Diet/CVRM (n=6)	6	11.0	0.5

In the Wilcoxon's test, the smaller the value of R to the critical value, the greater the difference between the two sets of measurements and the greater the likelihood that there is a significance difference between the pairs of data (Langley, 1971). The diet group (n=20) had an R of 63, with a critical value of 60; while the CVRM group (n=18) had an R value of 34.5, with a critical value of 40. Only the CVRM group (n=18) had an R low enough for the differences to be deemed significant at the 0.05 level.

A goodness-of-fit test (chi-square) was performed on the differences between the baseline and follow-up HDL ratios to determine if the sample came from a population with a normal distribution. It produced a chi-square value of 0.552. For one degree of freedom, 3.841 must be exceeded to establish significance at the .05 level; therefore, the assumption that the population is normally distributed was accepted (Crow, Davis & Maxfield, 1960).

Since the differences come from a population with a normal distribution for all samples, a one-way analysis of variance was done to compare changes in the CVRM and diet groups' baseline and follow-up HDL ratios. This procedure revealed no statistically significant differences between the two sample means (Table 4).

Table 4

Summary of Analysis of Variance of HDL Means (N=38)

Source	SS	df	MS	F	P
Between	1.562	1	1.562	.998	.3245
Within	56.372	36	1.566		
Total	57.935	37			

A regression analysis was performed on the relationship between changes in food frequency checklist scores and HDL ratios to answer the question: What is the relationship between dietary

changes reported on food frequency checklists and changes in HDL ratios? The analysis indicated no correlation (r= -0.01) between the two variables (Figure 1).

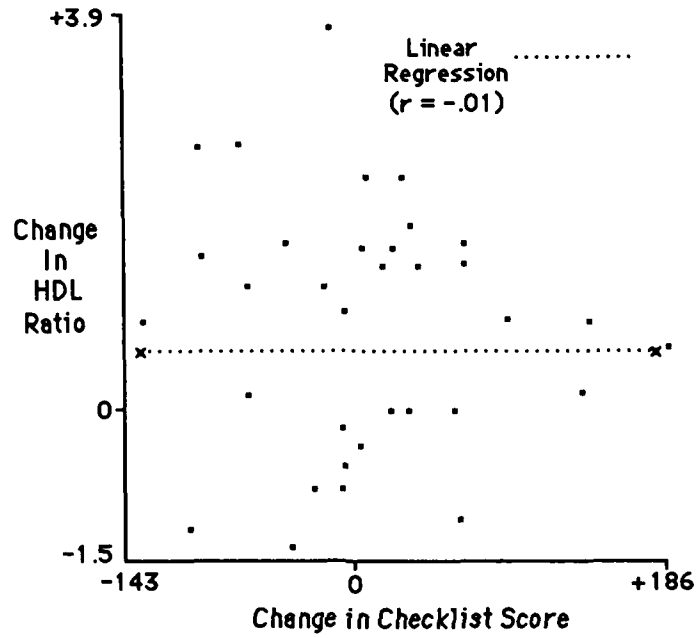

Figure 1
Scatter Plot of Changes in
Food Frequency Checklist Scores and HDL Ratios

Several problems were encountered in analyzing the results of the food frequency checklist. First, some of the variation in pre- and post-treatment scores may be attributed to the fact that the researcher was unable to personally instruct the participants on how to fill out the questionnaire. Second, before completing the baseline questionnaire, participants were told that if they never consumed an item they were to leave the line blank; a blank line was scored as zero. However, items consumed "rarely (less than one time a month)" were given a score--which, for some items was as much as 11. Thus, though there may have been little to no change in an individual's intake of an item over a 1-2 month period, just a slight change in his estimated consumption of an item

could change a total score by as much as 11 points--or 3 to 20% of the total. Using the tool in this way could account for some of the notable increases seen in post-treatment scores.

Discussion

This study's first question was what are the differences in the HDL ratios between participants in a CVRM program and a dietary cholesterol class? Data from this study did not show a significant difference between the two groups' mean HDL ratio differences. One factor affecting the results may have been the small sample size. Another may have been that the researcher did not assess the subjects' prior knowledge of dietary intake of cholesterol and saturated fats. Since a sample of convenience was used, this study's finding cannot be generalized beyond the group studied.

However, the CVRM group's baseline and follow-up mean HDL scores did demonstrate a significant difference which was not found in the diet group or its subgroups. This may reflect the efficacy of the CVRM program in providing participants with the knowledge and follow-up support needed to effect lifestyle changes.

There remains a question: How can there be a significant difference between the baseline and follow-up HDL ratio means of the CVRM group (and not the diet group) but no significant difference between the two groups? Although, at the .05 level, statistical significance can be shown for the CVRM group's mean change of 0.86 (but not the diet group's change of 0.45) the difference between the two groups (0.41) is smaller and, with standard deviations of 1.35 and 1.15, respectively, not statistically significant.

The finding that the CVRM program effected HDL ratio changes supports the results of previous studies. Whitney and Boswell (1986) showed that the CVRM program format used in this study was effective in bringing about lifestyle changes that significantly ($p<.001$) decreased the participants' HDL ratios. Other studies (Scalzi, Burke, & Greenland, 1980; Tirrell & Hart, 1980)

showed that the follow-up component of the CVRM program (missing in the diet group format) was an important variable in improving compliance to medical regimens.

CHAPTER FIVE

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

This investigation was conducted for the purpose of comparing the effectiveness of two types of preventive health education programs in bringing about dietary changes in individuals at high risk for cardiovascular disease. The questions explored were: What are the differences between changes in the HDL ratios of participants in a CVRM program and those in a dietary cholesterol class, and what is the relationship between dietary changes reported on food frequency checklists and changes in HDL ratios?

The sample consisted of 38 CVD high-risk males and females who received two different types of preventive health education. Their ages ranged from 20 to over 70. All participants had elevated HDL ratios on entry into the study and responded to a food frequency checklist. Six to eight weeks after intervention, a second HDL ratio was drawn and another food frequency checklist was completed by all participants.

A one-way analysis of variance was used to test for differences between the two types of programs. With the significance level at $p \leq .05$, no difference was shown. Additional analysis was conducted to explore differences between the baseline and follow-up HDL ratios of the two groups. A significant ($p=.05$) difference was found between the baseline and follow-up HDL means of the CVRM group. Regression analysis revealed no relationship between dietary changes reported on the food frequency checklists and changes in HDL ratios.

Conclusions

Findings of the study indicate that, for this sample, there were no significant ($p=.05$) differences in changes in HDL ratios between a group attending a CVRM program and a group

attending a cholesterol diet class. These findings, however, cannot be generalized to other populations at high risk for CVD.

Although no significant difference was noted between groups, the means of the CVRM baseline and follow-up HDL ratios indicated a significant improvement at the .05 level. This improvement was not found in either the diet group or its subgroups.

Limitations

Limitations of this study include:

1. A follow-up HDL ratio (measured after 6-8 weeks) does not necessarily reflect an individual's long-term dietary changes.

2. The researcher had no control over the choice of class instructors (whose instructional skills can affect the success of their program).

3. Because of the use of nonprobability sampling, the results cannot be generalized beyond this small sample.

4. Not all types of hyperlipidemia that elevate HDL ratios respond equally well to cholesterol/saturated fat diet modification.

5. No attempt was made to control for previous instruction in diet modification for cholesterol and saturated fat; therefore, there was no way of knowing the effects of any previous teaching.

Recommendations

Recommendations for further study are:

1. Replication of this study:
   a. using a larger sample;
   b. using a different diet survey to correlate dietary changes and HDL ratio changes;
   c. using one instructor for both groups;

d. controlling for previous knowledge concerning diet modification for cholesterol and staurated fat.

2. A 1-year longitudinal study to investigate the long term effects of a CVRM program, with testing at the 2, 6, and 12-month points.

BIBLIOGRAPHY

BIBLIOGRAPHY

Agruso, V. M. (1978). Learning in the later years: Principles of educational gerontology (p. 121). New York: Academic Press.

American Heart Association. (1987). Heart Facts Dallas, TX: Author.

Antischkow, N. (1933). Experimental arteriosclerosis in animals. In E. V. Cowdry (Ed.), Arteriosclerosis. New York: MacMillan Publishing.

Armstrong, J. L. (1976). Regression of atherosclerosis. In R. Paoletti & A. Coto (Eds.), Atherosclerosis (Vol. I). New York: Raven Press.

Armstrong, M. L., Warner, E. D., & Connor, W. E. (1970). Regression of atheromatosis in rhesus monkeys. Circulation Research, 27, 59.

Arnitzenius, A. C., Kromhout, D., Barth, J. D., Reiber, J. H. C., Bruschke, A. V. G., Buis, B., van Gent, C. M., Kempen-Voogd, N., Strikwerda, S., & Vander Velde, E. A. (1985). Diet, lipoproteins, and the progression of coronary atherosclerosis: The Leiden intervention trail. New England Journal of Medicine, 312 (13), 805-811.

Ayers, C., Allred, C., Short, J., Nowacek, G., Cox, D., Dewitt, C. (1985). Preventive cardiology, student manual. University of Virginia School of Medicine, Charlottesville, VA.

Barndt, R., Jr., Blankenhorn, D. H., Crawford, D. W., & Brooks S. H. (1977). Regression and progression of early femoral atherosclerosis in hyperlipoproteinemic patients. Annals of Internal Medicine, 86 (2), 139-146.

Becker, M. H., & Maiman, L. A. (1980). Strategies for enhancing patient compliance. Journal of Community Health, 6 (2), 113-135.

Becker, M. H., Maiman, L. A., Kirscht, J. P., Haefner, D. P., Drachman, R. H., & Taylor D. W. (1979). Patient perceptions and compliance: Recent studies of the health belief model. In R. B. Haynes, D. W. Taylor & D. Sackett (Eds.), Compliance in health care (pp. 78-109). Baltimore: John Hopkins University Press.

Behari-Varga, M., Szekely, J., & Gruber, E. (1981). Plasma high density lipoproteins in coronary, cerebral, and peripheral vascular disease. Atherosclerosis, 40, 337-345.

Carlson, L. A., & Bottiger, L. E. (1981). Serum triglycerides, to be or not to be a risk factor for ischemic heart disease? Atherosclerosis, 39, 287-291.

Castelli, W. P. (1985). Categorical issues in therapy for coronary heart disease. Cardiology in Practice, 3, (1), 267-273.

Castelli, W. P., Doyle, J. T., Gordon, T., Homes, C. G., Hjortland, M. C., Hulley, S. B., Kagan, A., & Zukel, W. J. (1977). HDL cholesterol and other lipids in coronary heart disease. Circulation, 55 (5), 767-772.

Chemistry Resource Committee, College of American Pathologists. (1988). Comprehensive chemistry 1987 survey: Author.

Cooper, R., & Van Horn, L. (1980). Nutrition and the coronary patient. In P. S. Fardy, J. L. Bennett, N. L. Reitz, & M. A. Williams (Eds.), Cardiac Rehabilitation (pp 174-193). St Louis: Mosby.

Crow, E. L., Davis, F. A., & Maxfield, M. W. (1960). Statistics Manual (pp. 87-90). New York: Dover Publications.

Dawber, T. R. (1980). The Framingham study: The epidemiology of atherosclerotic disease (pp. 121-141). Cambridge, MA.: Harvard University Press.

Dayton, S., Pearce, M. L., Hachimoto, S., Dixon, W. J., & Tomiyasu, U. (1969). A controlled clinical trial of a diet high in unsaturated fat in preventing complications of atherosclerosis. Circulation, 40 (Suppl. II), II-1 - II-63.

Eben, J. D., Nation, M. J., Marriner, A., & Nordmeyer, S. B. (1986). Dorothea E. Orem: Self-Care Deficit Theory of Nursing. In A. Marriner (Ed.), Nursing Theorists and Their Work (pp. 117-130). Princeton, NJ: C.V. Mosby.

Eder, H. A., & Gidez, L. I. (1982). The clinical significance of the plasma high density lipoproteins. Medical Clinics of North America, 66 (2), 431-440.

Enos, W. F., Holmes, R. H., & Beyer, J. (1953). Coronary disease among United States soldiers killed in action in Korea. Journal of the American Medical Association, 152, 1090-1093.

Gordon, T., Castelli, W. P., Hjortland, M. C., Kannel, W. B., & Dawber, T. R. (1977). High density lipoprotein as a protective factor against coronary heart disease. American Journal of Medicine, 62, 707-714.

Green, L. W. (1979). Educational strategies to improve compliance with therapeutic and preventive regimens: The recent evidence. In R. B. Haynes, D. W. Taylor & D. Sackett (Eds.), Compliance in health care (pp. 157-173). Baltimore: John Hopkins University Press.

Grundy, S. M., Bilheimer, D., Blackburn, H., Brown W. V., Kwiterovich, P. O., Mattson, F., Schonfeld, G., & Weidman, W. H. (1982). Rationale of the diet-heart statement of the American Heart Association: Report of nutrition committee. Circulation, 65 (4), 839A-854A.

Haynes, C. (1984). Primary intervention for pain due to inadequate myocardial perfusion. In D. Sadler (Ed.), Nursing for cardiovascular health, (pp. 189-235). East Norwalk, CT: Appleton-Century-Crofts.

Hjermann, I., Velve Byre, K., Holme, I., & Leren, P. (1981). Effect of diet and smoking intervention on the incidence of coronary heart disease: Report from the Oslo study group of a randomized trial of healthy men. Lancet, 2 (8259). 1301-1310.

Kannel, W. B. (1976). Some lessons in cardiovascular epidemiology from Framingham. American Journal of Cardiology, 37(2), 269-282,

Kannel, W. B. (1986). Epidemiologic insights into atherosclerotic cardiovascular disease from the Framingham study. In M. L. Pollock & D. H. Schmidt (Eds.), Heart Disease and Rehabilitation (2nd ed.) (pp.13-26). New York: Wiley & Sons.

Kannel, W. B., Castelli, W. P., & Gordon, T. (1979). Cholesterol in the prediction of atherosclerotic disease. Annals of Internal Medicine, 90 (1), 85-91.

Kannel, W. B., Castelli, W. P., Gordon, T., & McNamara, P. M. (1971). Serum cholesterol, lipoproteins, and the risk of coronary heart disease: The Framingham study. Annals of Internal Medicine, 74 (1), 1-12.

Kannel, W. B., Dawber, T. R., Kagan, A., Revotskie, N., & Stokes, J. (1961). Factors of risk in the development of coronary heart disease: Six-year follow-up experience, the Framingham study. Annals of Internal Medicine, 55 (1), 33-50.

Katz, L. N., & Stamler, J. (1953). Experimental arteriosclerosis. Springfield, IL: Charles C. Thomas.

Keys, A. (Ed.). (1970). Coronary heart disease in seven countries. Circulation, 41 (Suppl. I), I-1 - I-190

Keys, A., Kimura, N., Kusukawa, A., Bronte-Stewart, B., Larsen, N., & Keys, M. H. (1958). Lessons from serum cholesterol studies in Japan, Hawaii and Los Angeles. Annals of Internal Medicine, 48 (1), 83-94.

Kinlein, M. L. (1977). The self-care concept. American Journal of Nursing, 77, 598-601.

Knowles, M. (1978). The adult learner: A neglected species (2nd ed.), (pp. 55-58). Houston, TX: Gulf Publishing.

Knowles, M. (1980). The modern practice of adult education: From pedagogy to andragogy, (pp. 41-42). Chicago: Association Press, Follett Publishing.

Kornitzer, M., DeBackes, G., Dramaix, M., & Thilly, C. (1980). The Belgian heart disease prevention project: Modification of the coronary risk profile in an industrial population. Circulation, 61 (1), 18-25.

Kushi, L. H., Lew, R. A., Stare, F. J., Ellison, C. R., el Lozy, M., Bourke, G., Daly, L., Graham, I., Hickey, N., Mulcahy, R., & Kevaney, J. (1985). Diet and 20-year mortality from coronary heart disease, the Ireland-Boston diet-heart study. New England Journal of Medicine, 312, (13), 811-818.

Laboratory Standardization Panel of the National Cholesterol Education Program. (1988). Current status of blood cholesterol measurement in clinical laboratories in the United States. Laboratory Medicine, 19 (5), 324-331.

Langhans, P. A. (1984). Underlying causes of pain due to inadequate myocardial perfusion. In D. Sadler, (Ed.), Nursing for cardiovascular health, (pp. 167-171). East Norwalk, CT: Appleton-Century-Crofts.

Langley, R. (1971). Practical statistics simply explained (pp. 166-187). New York: Dover Publications.

Leren, P., Helgeland, A., Hjermann, I., & Holme, I. (1983). MRFIT and the Oslo study. Journal of the American Medical Association, 249 (7), 893-894.

Levy, R. I. (1986). Changing perspectives in the prevention of coronary artery disease. American Journal of Cardiology, 57, 17G-26G.

Levy, R. I., Brensike, J. F., Epstein, S., Kelsey, S., Passamani, E. R., Richardson, J. M., Loh, I. K., Stone, N. J., Aldrich, R. F., Battatlini, J. W., Morearty, D. J., Fisher, M. L., & Detre, K. M. (1984). Influence of changes in lipid values induced by cholestyramine and diet on progression of coronary artery disease: Results of the NHLBI type II coronary intervention study. Circulation, 69, 325-337.

Lipid Research Clinics' Program. (1984). The lipid research cinics' coronary primary prevention trial results: Reduction in evidence of coronary heart disease. Journal of the American Medical Association, 251 (3), 351-364.

Lovvorn, J. (1980). Types of preventive health cues given to high-risk individuals. Heart & Lung, 10 (3), 520-524.

Malinow, M. R. (1981). Regression of atherosclerosis in humans: Fact or myth. Circulation, 64 (1), 1-3.

McGill, H. C., Jr. (Ed.). (1968). The Geographic Pathology of Atherosclerosis, (p. 41). Baltimore: Williams & Wilkins.

McKeigue, P. M., Marmot, M. G., Adelstein, A. M., Hunt, S. P., Shipley, M. J., Butler, S. M., Riemersma, R. A., & Turner, P. R. (1985). Lancet, 2 (8464), 1086-1090.

McNamara, J. J., Molot, M. A., Stremple, J. F., & Cutting, R. T. (1971). Coronary artery disease in combat casualties in Viet Nam. Journal of the American Medical Association, 216 (7), 1185-1187.

Miettinen, M., Turpeinin, O., Karvonen, M. J., Elosuo, R., & Paavilainen, E. (1972). Effect of cholesterol-lowering diet on mortality from coronary heart-disease and other causes. Twelve-year clinical trial in men and women. Lancet, 2 (7782), 835-838.

Miller, G. J., & Miller N. E. (1975). Plasma-high-density-lipoprotein concentration and development of ischaemic heart-disease. Lancet, 1 (7897), 16-19.

Miller, N. E. (1982). Coronary atherosclerosis and plasma lipoproteins: Epidemiology and pathophysiologic considerations. Journal of Cardiovascular Pharmacology, 4 (Suppl. 2), S190-S195.

Moberg, B., & Wallentin, L. (1981). High density lipoprotein and other lipoproteins in normolipidaemic and hypertriglyceridaemic (type IV) men with coronary artery disease. European Journal of Clinical Investigation, 11 (6), 433-440.

Muhlenkamp A., & Sayles, J. (1986). Self esteem, social support, and positive health practices. Nursing Research, 35, 334-338.

Multiple Risk Factor Intervention Trial Research Group. (1982). Multiple risk factor intervention trial: Risk factor changes and mortality results. Journal of the American Medical Association, 248 (12), 1465-1477.

National Cholesterol Education Program. (1988). Report of the National Cholesterol Education Program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. Archives of Internal Medicine, 148, 36-69.

Oslo Study Research Group. MRFIT and the Oslo study. Journal of the American Medical Association, 249 (7), 893-894.

Page, L. B., Damon, A., & Moellering, R. C., Jr. (1974). Antecedents of cardiovascular disease in six Solomon Islands' societies. Circulation, 49, 1132-1145.

Pender, N., & Pender, A. (1986). Attitudes, subjective norms and intentions to engage in health behaviors. Nursing Research, 35, 15-18.

Pooling Project Research Group. (1978). Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: Final report of the Pooling Project. Journal of Chronic Disease , 31 (4) (Spec. Iss.), 201-306.

Rhoads, G. G., Gulbrandsen, C. L., & Kagan, A. (1976). Serum lipoproteins and coronary heart disease in a population study of Hawaii Japanese men. New England Journal of Medicine, 294 (6), 293-298.

Rosenman, R. H., Brand, R. J., Sholtz, R. I., & Friedman, M. (1976). Multivariate prediction of coronary heart disease during 8.5 year follow-up in the Western Collaborative Group study. American Journal of Cardiology, 37, 903-910.

Rossner, S., Kjellin, K. G., Mettinger, K. L., Siden, A., & Soderstrom, C. E. (1978). Dyslipoproteinemia in patients with ischemic cerebro-vascular disease. Atherosclerosis, 30 (3), 199-209.

Scalizi, C. C., Burke, L. E., & Greenland, S. (1980). Evaluation of an inpatient education program for coronary patients and families. Heart & Lung, 9, 846-853.

Shekelle, R. B., Shryock, A. M., Paul, O., Lepper, M., Stamler, J., Lie, S., & Raynor, W. J. (1981). Diet, serum cholesterol, and death from coronary heart disease: The Western Electric study. New England Journal of Medicine, 304 (2), 65-70.

Simons, L. A. (1986). Interrelations of lipids and lipoproteins with coronary artery disease mortality in 19 countries. American Journal of Cardiology, 57, 5G-10G.

Stamler, J. (1978). Lifestyles, major risk factors, proof and public policy. Circulation, 58 (1), 3-19.

Strong, J. P., & McGill, H. C., Jr. (1967). Diet and experimental atherosclerosis in baboons. American Journal of Pathology, 50, 669.

Tirrell, B., & Hart, L. (1980). The relationship of health beliefs and knowledge to exercise compliance in patients after coronary bypass. Heart & Lung, 9, 487-493.

Vesselinovitch, D., Wissler, R. W., Hughes, R., & Borensztajn, J. (1976). Reversal of advanced atherosclerosis in rhesus monkeys. Atherosclerosis, 23, 155-76.

Vogelberg, K. H., Berchtold, P., Berger, H., Gries, F. A., Klinger, H., Kubler, W, & Stolze T. H. (1975). Primary hyperlipoproteinemias as risk factors in peripheral artery disease documented by arteriography. Atherosclerosis, 22, 271-285.

Webster's Ninth New Collegiate Dictionary. (1986). Springfield, MA.: Merriam-Webster.

West, R. O., & Hayes, O. B. (1964). Diet and serum cholesterol levels --a comparison between vegetarians and nonvegetarians in a Seventh-Day Adventist group. American Journal of Clinical Nutrition, 21, 853.

Whitney, E. J., & Boswell, R. N. (1986). Cardiovascular risk modification: A multidisciplinary approach in a USAF clinic setting. Military Medicine, 151 (9), 473-477.

Wissler, R. W. (1974). Development of the arteriosclerotic plaque. In E. Braunwald (Ed.), Myocardium: Failure and infarction. New York: H. P. Publishing.

World Health Organization, Committee of Principle Investigators. (1984). WHO cooperative trial in primary prevention of ischemic heart disease with clofibrate to lower serum cholesterol: Final mortality follow-up. Lancet, 2, (8403),600-604.

Worth, R. M., Kato, H., Rhoads, G. G., Kagan, A., & Syme, S. L. (1975). Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: Mortality. American Journal of Epidemiology, 102, 481.

APPENDIX A

FOOD FREQUENCY CHECKLIST

FOOD FREQUENCY CHECKLIST

Name_____ Date_____

Check how often you eat the following foods:

	Rarely (less than 1 per month)	1-2 times per month	1-2 times per week	3-7 times per week	More than 1 per day
MEAT, FISH & POULTRY					
Regular ground beef (M)*	Û	Û	Û	Û	Û
Lean ground beef (L)*	Û	Û	Û	Û	Û
Sausage, bacon, hot dogs, luncheon meat (H)*	Û	Û	Û	Û	Û
Poultry with skin (chicken, turkey, etc.) (H)	Û	Û	Û	Û	Û
Poultry w/o skin (chicken, turkey, etc.) (L)	Û	Û	Û	Û	Û
Fish (L)	Û	Û	Û	Û	Û
Shrimp (M)	Û	Û	Û	Û	Û
Shellfish (crab, scallops, oysters, etc.) (L)	Û	Û	Û	Û	Û
Organ meats (liver, heart, brains, etc.) (H)	Û	Û	Û	Û	Û
Eggs (H)	Û	Û	Û	Û	Û
DAIRY PRODUCTS					
Whole milk (H)	Û	Û	Û	Û	Û
Non-dairy creamer (M)	Û	Û	Û	Û	Û
Cream, half & half (H)	Û	Û	Û	Û	Û
Lowfat milk (M)	Û	Û	Û	Û	Û
Nonfat milk (L)	Û	Û	Û	Û	Û
Cheese, low fat (L)	Û	Û	Û	Û	Û
Hard cheese, cheese spread, regular cottage cheese (H)	Û	Û	Û	Û	Û
Ice cream (H)	Û	Û	Û	Û	Û
Sherbert, Ice milk (L)	Û	Û	Û	Û	Û
Whipped cream (H)	Û	Û	Û	Û	Û
Non-dairy whipped topping (M)	Û	Û	Û	Û	Û

	Rarely (less than 1 per month)	1-2 times per month	1-2 times per week	3-7 times per week	More than 1 per day
FATS					
Oils (in cooking, etc.) (L)					
Kind_____	Û	Û	Û	Û	Û
Butter (H)	Û	Û	Û	Û	Û
Margarine, list first ingredient on label (L) _____	Û	Û	Û	Û	Û
Salad dressing (L) Kind_____	Û	Û	Û	Û	Û
Peanut butter (L)	Û	Û	Û	Û	Û
Nuts and seeds (L)	Û	Û	Û	Û	Û
Fried meats or vegetables (M)	Û	Û	Û	Û	Û
BREADS AND GRAINS					
Breads, white or whole grain (L)	Û	Û	Û	Û	Û
Quick breads, commercial (M) (biscuits, muffins,pancakes, waffles, etc.)	Û	Û	Û	Û	Û
Quick breads, homemade (M) (biscuits, muffins, pancakes, waffles, etc.)	Û	Û	Û	Û	Û
Cereals (L)	Û	Û	Û	Û	Û
Pasta (noodles, rice, etc.) (L)	Û	Û	Û	Û	Û
Commercially-baked goods (H) (cookies, pies, cakes, etc.)	Û	Û	Û	Û	Û
Homemade-baked goods (M)	Û	Û	Û	Û	Û
Snack crackers,(L/M/H) Kind_____	Û	Û	Û	Û	Û
VEGETABLES					
Vegetables (0)	Û	Û	Û	Û	Û
Beans (navy, soy, blackeyed peas, bean & pea soups, etc.) (0)	Û	Û	Û	Û	Û

	Rarely (less than 1 per month)	1-2 times per month	1-2 times per week	3-7 times per week	More than 1 per day
FRUITS					
Fruit (0)	Û	Û	Û	Û	Û
Fruit juice (0)	Û	Û	Û	Û	Û
SNACKS					
Potato chips, corn chips, etc. (M)	Û	Û	Û	Û	Û
Candy (M)	Û	Û	Û	Û	Û
ETHNIC					
Chinese (L)	Û	Û	Û	Û	Û
Italian (M)	Û	Û	Û	Û	Û
Mexican (M)	Û	Û	Û	Û	Û
Soul (H)	Û	Û	Û	Û	Û

*Weighted for cholesterol/saturated fat content.
L = Low (scored 1-5)
M = Medium (scored 6-10)
H = High (scored 11-15)

APPENDIX B

PERMISSION LETTER

DEPARTMENT OF THE AIR FORCE

1 MEDICAL GROUP (TAC)
LANGLEY AIR FORCE BASE VA 23665-5300

51 A

REPLY TO ATTN OF: SGH (Col Collier, 46485)

17 FEB 1988

SUBJECT: Approval of Data Collection for a Thesis

TO: Major Lois Schwartz
102 White House Drive
Poquoson, VA 23662

I am pleased to inform you that Colonel Gross, 1st Medical Group Commander, has approved your request to collect data at the 1st Medical Group for your thesis comparing two education programs for reducing cardiovascular risk. Good luck with your thesis and if I can be of any further assistance, please do not hesitate to call me.

JAMES R. COLLIER, Colonel, USAF, MC
Chief, Hospital Services

Providing the Reins of Command

APPENDIX C

INFORMED CONSENT FORM (DIETARY GROUP)

Informed Consent - Diet Group

I am Major Lois Schwartz, an Air Force Institute of Technology (AFIT) graduate student studying for a masters degree in nursing at the VCU/MCV School of Nursing in Richmond. As part of my requirements, I am conducting a study to explore the differences between two methods of instruction on factors which reduce the risk of developing heart disease, and the subsequent improved health behaviors. People who participate in this study will be taught health practices that, if applied, could help them live longer, healthier lives. This study program has been approved by the Commander of the Langley AFB Hospital.

If you agree to participate in the study program you will:

1. Complete a fact sheet asking your age, sex, marital status, education level, and personal and family history of heart disease.

2. Complete a questionnaire about how often you eat different types of foods (prior to attending class).

3. Attend the one-hour class on low fat/low cholesterol diet.

4. Complete a questionnaire 6-8 weeks after attending the class on your frequency of intake of different foods.

Participation in this study program is voluntary and, whether or not you agree to participate, will in no way affect your health care at Langley AFB Hospital. Neither will your participation in the study program present any risk to your health. You may withdraw from the study at any time by contacting me through my faculty advisor, whose name and telephone number are at the bottom of this form. No participant will be identified by name in any report about the study program; only group information will be reported.

If you have any questions about this study, the class instructor will give instructions on how to contact me. Please do not hesitate to call.

If you agree to participate in the study, give the signed consent to the instructor and keep a copy for your records. Thank you for considering this request.

I have read the above information about the health behavior study being conducted at Langley AFB Hospital and I agree to participate in the program.

_____ _____

Signature Date

If you decide to withdraw from the program, please contact me through:

Dr. M. Olgas

Associate Professor of Nursing

Telephone: 1-786-0707

APPENDIX D

INFORMED CONSENT FORM (CVRM GROUP)

Informed Consent - CVRM Group

I am Major Lois Schwartz, an Air Force Institute of Technology (AFIT) graduate student studying for a masters degree in nursing at the VCU/MCV School of Nursing in Richmond. As part of my requirements, I am conducting a study to explore the differences between two methods of instruction on factors which reduce the risk of developing heart disease, and the subsequent improved health behaviors. People who participate in this study will be taught health practices that, if applied, could help them live longer, healthier lives. This study program has been approved by the Commander of the Langley AFB Hospital.

If you agree to participate in the study program you will:

1. Complete a fact sheet asking your age, sex, marital status, education level, and personal and family history of heart disease.

2. Complete a questionnaire about how often you eat different types of foods (prior to attending class).

3. Attend both of the two and a half hour classes on the anatomy and physiology of the heart, disease of the heart and blood vessels, risk factors of heart disease, and recommended changes to reduce your risk.

4. Complete a questionnaire 6-8 weeks after attending the class on your frequency of intake of different foods.

Participation in this study program is voluntary and, whether or not you agree to participate, will in no way affect your health care at Langley AFB Hospital. Neither will your participation in the study program present any risk to your health. You may withdraw from the study at any time by contacting me through my faculty advisor, whose name and telephone number are at the bottom of this form. No participant will be identified by name in any report about the study program; only group information will be reported.

If you have any questions about this study, the class instructor will give instructions on how to contact me. Please do not hesitate to call.

If you agree to participate in the study, give the signed consent to the instructor and keep a copy for your records. Thank you for considering this request.

I have read the above information about the health behavior study being conducted at Langley AFB Hospital and I agree to participate in the program.

_____ _____

Signature Date

If you decide to withdraw from the program, please contact me through:

Dr. M. Olgas

Associate Professor of Nursing

Telephone: 1-786-0707

APPENDIX E

DIETARY CLASS CONTENT

WARNING: THIS DIET SHOULD NOT BE USED WITHOUT A PRESCRIPTION FROM A PHYSICIAN OR CREDENTIALED HEALTH CARE PROVIDER.

This diet instruction has been given to you

by _____,
(Name of health care provider)

at _____,
(Name of medical facility)

on _____.
(Date)

If questions arise, direct them to the health care

provider at _____.
(Telephone number)

DEPARTMENT OF THE AIR FORCE
Headquarters US Air Force
Washington DC 20330

AF PAMPHLET 166-42

14 December 1983

Medical Food Service

FAT CONTROLLED, CHOLESTEROL RESTRICTED DIET

This pamphlet provides guidance needed by the patient to follow the diet prescribed by a physician or other health care provider. It also provides instructions to meet the nutritional requirements of each individual within the restrictions imposed by the diet. The use of a name of any specific manufacturer, commercial product, commodity, or service in this publication does not imply endorsement by the Air Force.

Principles

A diet that is restricted in cholesterol and saturated fat can help to lower blood cholesterol levels.

Introduction

This booklet provides information to help you plan and select a well-balanced, nutritionally adequate diet. The following is general information about cholesterol and saturated fat.

Cholesterol and Saturated Fat

The usual daily intake of cholesterol is 600 to 900 milligrams. This diet moderately restricts dietary cholesterol to 300 to 500 milligrams a day. Cholesterol and saturated fat are controlled to reduce blood cholesterol levels. Blood cholesterol has two sources: the cholesterol in the diet and that made in the body, mainly in the liver. There are three major dietary changes that will lower cholesterol in most people. These are:

(1) Restriction of dietary cholesterol.
(2) Restriction of saturated fat.
(3) Increased intake of polyunsaturated fat.

Dietary Cholesterol is found only in foods of animal origin. Egg yolk and organ meats are very high in cholesterol. Shrimp is moderately high. Any food containing meat or meat fat has cholesterol. Foods of plant origin contain no cholesterol.

Supersedes AFP 160-160, 19 June 1981.
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Approved by: Col N. C. Nicholas
Writer-Editor: Novella S. Hill
Distribution X (medical units only)

Saturated Fat is usually a fat of animal origin. It tends to raise the blood cholesterol and is restricted in this diet. Saturated fat is in such foods as butter, cream, whole milk, and cheese made from whole milk or cream. Among the few vegetable fats that are saturated are coconut oil and palm oil (used in nondairy cream substitutes and some frozen desserts) and cocoa butter (the fat in chocolate). Other vegetable oils, unsaturated by nature, can be made saturated by hardening or hydrogenation. The amount of saturation is indicated by the degree of hardness. For example, a tub of margarine is less saturated than a stick of margarine.

Polyunsaturated Fats are fats of plant origin. They are thought to help lower blood cholesterol. Vegetable oils vary in the degree of unsaturation. Safflower oil is, for example, more unsaturated than olive oil.

Cholesterol Content of Common Foods

Food	Amount	Cholesterol (mg.)
Milk, whole	1 c.	34
Milk, lowfat, 1% fat	1 c.	14
Milk, nonfat, fluid or reconstituted dry	1 c.	5
Yogurt, lowfat fruited	8 oz.	13
Yogurt, skim milk	8 oz.	5
Cream, whipping	1 fl. oz.	40
Cream, half and half	¼ c.	26
Cream, light table	1 fl. oz.	20
Cream, cheese	1 tbsp.	16
Cream, sour	2 tbsp.	16
Frozen custard or French ice cream	½ c.	48
Ice cream, regular, approximately 14% fat	½ c.	43
Ice cream, regular, approximately 10% fat	½ c.	27
Cheese:		
Gouda or Gruyere	1 oz.	33
Brick, mild or sharp Cheddar, or Romano	1 oz.	31
Bleu, Colby, Muenster, pasteurized processed American or Swiss, Roquefort, Swiss	1 oz.	28
Camembert, Edam, or Feta	1 oz.	26
Cottage cheese, creamed, 4% fat	½ c.	24
Grated Parmesan	1 oz.	24
Provolone, American cheese food	1 oz.	20
American cheese spread, part-skim low moisture Mozzarella	1 oz.	16
Cottage cheese, creamed, 1% fat	½ c.	12

Food	Amount	Cholesterol (mg.)
Ricotta, part skim	1 oz.	9
Cottage cheese, uncreamed	½ c.	7
Clams, halibut, tuna	3 oz. cooked	55
Oysters, salmon	3 oz. cooked	40
Crab, lamb, veal	3 oz. cooked	85
Beef, chicken or turkey (dark meat), lobster, pork	3 oz. cooked	75
Chicken or turkey (light meat)	3 oz. cooked	67
Egg	1 yolk or 1 egg	250
Heart, beef	3 oz. cooked	230
Shrimp	3 oz. cooked	130
Brains	3 oz. raw	>1700
Kidney	3 oz. cooked	680
Liver, beef, calf, hog, lamb	3 oz. cooked	370
Butter	1 tbsp.	35
Lard	1 tbsp.	12

In making up your diet plan, the foods have been selected from four basic food groups—(1) milk, (2) meat (and other protein foods), (3) fruits and vegetables, and (4) breads (and other grain products).

When these foods are taken as recommended in your "Food Suggestion List" and the "Guide to Good Eating,*" they will supply ample amounts of protein, carbohydrate, fat, vitamins, and minerals necessary for your good health.

Guide to Good Eating

A Recommended Daily Pattern

Food Group	Recommended Number of Servings				
	Child	Teenager	Adult	Pregnant Woman	Lactating Woman
Milk 1 cup milk, yogurt OR Calcium Equivalent: 1½ slices (1½ oz) cheddar cheese* 1 cup pudding 1 3/4 cups ice cream 2 cups cottage cheese*	3	4	2	4	4
Meat 2 ounces cooked, lean meat, fish, poultry, OR Protein Equivalent: 2 eggs 2 slices (2 oz) cheddar cheese* ½ cup cottage cheese* 1 cup dried beans, peas 4 tbsp peanut butter	2	2	2	3	2
Fruit—Vegetable ½ cup cooked or juice 1 cup raw Portion commonly served such as a medium-size apple or banana	4	4	4	4	4
Grain—whole grain, fortified, enriched 1 slice bread ½ cup cooked cereal 1 cup ready-to-eat cereal pasta, grits	4	4	4	4	4

*Count cheese as serving of milk OR meat, not both simultaneously.

"Others" complement but do not replace foods from the Four Food Groups. Amounts should be determined by individual caloric needs.

*Courtesy of the National Dairy Council.

Food Suggestions

Allowed	Avoid
Milk	
Buttermilk prepared with nonfat milk; nonfat milk	Buttermilk prepared with whole milk; chocolate milk, lowfat milk, whole milk
Cocoa prepared with nonfat milk and cocoa powder	Condensed or evaporated milk prepared with whole milk
Dried nonfat milk	Dried whole milk
Evaporated nonfat milk	Yogurt prepared with whole milk
Yogurt prepared with nonfat milk	
Egg	
Egg substitute	More than 3 whole eggs per week
Egg whites	
Whole eggs (3 per week)	
Meat or Substitute	
Limit lean beef, ham, lamb or pork to 3 oz.—3 times per week. Other meals to include no more than 6 oz. per day of clams, crab, fish, lobster, oysters, poultry, scallops, and veal.	
Trim all fat—	
Baked, boiled, broiled, grilled, roasted, or stewed:	
Lean beef, ham, lamb, liver (limit to one 3 oz. serving per week), pork, veal	Canned meats and meat mixtures
Without skin: chicken, Cornish game hen, game birds, turkey	Commercially fried meat, fish, or poultry
Baked, boiled, broiled, canned, dried, fresh, or poached:	Duck, goose, poultry skin
Fish, shellfish (Limit shrimp to two 4 oz. servings per week)	Fatty meats such as Canadian bacon, corned beef, frankfurters, heavily marbled meat, luncheon meat, regular hamburger, salt pork, sausage, spareribs.
	Frozen packaged casseroles or dinners
	Organ meats including brains, heart, kidney, liver (more than one 3 oz. serving per week), sweetbreads
	Shrimp (more than two 4 oz. servings per week)

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Allowed	Avoid
Cheese substitutes prepared with polyunsaturated oil	Cheese foods and spreads
Nonfat milk cheese, such as Baker's, dry cottage cheese, Farmer's, Hoop, Mozzarella, Sapsago	Cheese prepared with whole milk: American, Bleu, Brick, Camembert, Cheddar, Edam, Gouda, Neufchatel, Parmesan, Ricotta, Roquefort
Peanut butter	
Potato or Substitute	
Prepared with allowed ingredients:	
Barley	Egg noodles
Bread stuffing	Pork and beans
Macaroni, noodles, spaghetti, and other types of pasta	Any prepared with bacon, butter, cheese, cream, eggs, meat drippings, shortening, whole milk, or other foods not allowed
Rice	
Sweet or white potatoes, yams	
Wild rice	
Bread and Cereal	
Prepared with allowed ingredients—nonfat milk, egg allowance, polyunsaturated vegetable oil, or a minimum of saturated fat:	
Biscuits, cornbread, English muffins, French toast, hard rolls, muffins, pancakes, pan rolls, quick breads, tortillas, waffles, whole wheat rolls	Butter or cheese crackers
	Cereals containing coconut or fat
	Cheese or egg bread
Boston brown, cracked wheat, French, Italian, pumpernickel, raisin, rye, white, or whole wheat bread	Commercial biscuits, French toast, muffins, pancakes, pastries, sweet rolls, waffles
	Fried snack foods
	Popovers
	Spoon bread
Bread sticks, rusk, zwieback	
Cereals not on the avoid list	
Crackers not on the avoid list	
Vegetable	
All not on the avoid list	Any prepared with foods not allowed
Fruit	
All	None
Fat	
Corn, cottonseed, safflower, sesame, soybean, or sunflower oil	Bacon

Allowed	Avoid
French or Italian salad dressings, mayonnaise	Blue cheese, hydrogenated, and Roquefort salad dressings
Margarines with liquid vegetable oil as first ingredient	Butter, cream cheese, half and half, light cream, sour cream, whipping cream
Nondairy creamers prepared with unsaturated oil	Coconut and palm oil
	Gravy and sauces prepared with foods not allowed
	Hydrogenated margarines and shortenings
	Lard, suet
	Meat drippings
	Nondairy creamers prepared with saturated fat
Soup	
Bouillon, broth, consommé	Any prepared with butter, cheese, cream, eggs, whole milk, or other foods not allowed
Campbells:	
Black Bean	
Chicken Gumbo	
Chicken Vegetable	
Chicken with Rice	
Cream of Asparagus	
Cream of Potato	
Green Pea	
Vegetable	
Vegetable Beef	
Heinz:	
Beef Barley	
Beef Vegetable	
Chicken Gumbo	
Cream of Green Pea	
French Onion	
New England Style Clam Chowder	
Tomato	
Vegetable with Beef Broth	
Vegetarian Vegetable	
Packaged dried soup (broth-based)	
Any soups prepared with allowed ingredients	

Allowed	Avoid
Sweets	
Hard candy Honey, molasses, syrup Jam, jelly, and preserves Marshmallows Sugar	Any sweets prepared with butter, chocolate, coconut, cream, eggs, hydrogenated margarine or shortening, whole milk, or other foods not allowed.
Desserts	
Angel food cake, sponge cake Cakes, cobblers, cookies, frozen desserts, pastries, pies, puddings, sherbet prepared with nonfat milk, egg allowance, and polyunsaturated vegetable oil Fig bars, ginger snaps, sugar wafers Fruit whip, flavored gelatin, water-based ices	Cakes, cookies, frozen desserts, pastries, pies, puddings prepared with butter, chocolate, coconut, cheese, cream, eggs, hydrogenated margarine or shortening, whole milk or other foods not allowed
Beverages	
Carbonated beverages, cereal beverages, coffee, decaffeinated coffee, fruit flavored beverages, tea	Any beverages prepared with chocolate, cream, eggs, whole milk, or other foods not allowed
Miscellaneous	
All herbs and spices Baking powder and soda Barbecue, chili, cocktail, sweet-sour, tomato, soy, Worcestershire sauce Catsup, chutney, horseradish, mustard Cocoa powder Cream of tartar Extracts and flavorings Peanuts, pecans, walnuts Pepper Pickles Popcorn, pretzels Salt Vinegar Yeast	Cashews, coconut, macadamia nuts

Menu Pattern*

Breakfast	Dinner	Supper
Juice or fruit Cereal F/R egg (1) (3 times a week) or substitute Toast or breakfast bread F/C margarine (2) Jam or jelly Nonfat milk Coffee or decaffeinated coffee with F/C cream substitute, or hot or iced tea with lemon	F/C poultry, fish, or veal (3 oz.) F/C potato or substitute F/C vegetable F/C salad F/C salad dressing Bread or roll F/C dessert or fruit Nonfat milk Coffee or decaffeinated coffee with F/C cream substitute, or hot or iced tea with lemon	F/C meat (3 oz.) (3 times a week) F/C poultry, fish, or veal (3 oz.) (4 times a week) F/C potato or substitute F/C vegetable F/C salad F/C salad dressing F/C margarine (2) F/C dessert or fruit Nonfat milk Coffee or decaffeinated coffee with F/C cream substitute, or hot or iced tea with lemon

Sample Menu

Breakfast	Dinner	Supper
Orange juice Oatmeal Scrambled egg Toast Corn oil margarine Strawberry jam Nonfat milk Coffee	Tuna salad sandwich: Tuna fish (water packed) and mayonnaise on whole wheat bread Carrot and celery sticks Fresh apple Iced tea	Broiled veal steak Baked potato with nonfat yogurt and chives Green beans Lettuce and tomato salad Vinegar and oil dressing Hard roll Corn oil margarine Angel food cake Nonfat milk Coffee

*NOTE: F/C means fat controlled. F/R means fat restricted.

B12 + 13 not available

Vegetable Cookery

Vegetables, which are relatively free of all fat but are good sources of vitamins and minerals, play an important role in the diet. They should be well prepared and interesting. Adding margarine to them improves the flavor.

Combining two or more vegetables makes each seem different. A little onion cooked with green beans, squash, or peas picks up their flavor. Some fresh dill on carrots and basil or oregano in tomatoes changes them completely. Mashed potatoes with orange juice or chicken broth instead of milk makes them unusual and also eliminates the cholesterol of the milk.

Vegetables contain large amounts of minerals and vitamins as well as protein. To retain the most nutrients they should be cooked only until tender in as little water as possible. Steaming or sautéeing vegetables is highly recommended.

A very buttery flavor can be obtained from a small amount of margarine if vegetables are sautéed in the margarine with a very small amount of water. Prepared this way vegetables generally remain more crisp than when boiled.

Baking and broiling vegetables also retains more vitamins and minerals than boiling them. Frozen vegetables are especially good when baked in a covered casserole with a little margarine and small amount of salt. Since no water is added, use very little salt.

Fresh vegetables should be well washed before cooking but should not be allowed to soak in water. Often washing vegetables in tepid water and then storing them in the refrigerator will bring back their crispness.

Canned vegetables should be heated just until hot in the liquid from the can.

Breads, Cakes, and Cookies

Most bread, cake, and cookie recipes can be adapted to this diet simply by substituting margarine for the fat, skim milk for the whole milk, and egg substitute for the whole eggs.

Suggestions for Meals Away From Home

When you eat out daily, it is wise to select one particular restaurant. Some of your food requires special preparation. A "regular" customer is more likely to receive special attention.

Meat. Ask that all fat be trimmed. The safest choice is a chop, steak, chicken, or fish. Request the meat be broiled *without fat*. Limit the amount and kind of meat according to your diet plan.

Vegetable. Any vegetable prepared without fat may be included. None of those that are creamed or prepared in a sauce containing fat, whole milk, or cheese may be used.

Salad. Most ingredients such as vegetables, fruit, or gelatin are allowed on your diet. Avoid cheese, including cream cheese, sour cream, or whipped cream. You may select dressings such as Italian, French, or oil and vinegar if the waiter indicates that an oil allowed on your diet has been used. Often lemon or vinegar is the wisest choice.

Fat. Margarine, salad dressing, etc., that is listed in your diet may be selected.

Bread. Saltines are usually available; in addition, you may include plain sliced bread and hard rolls. Avoid hot rolls, biscuits, cornbread, popovers, muffins, etc., because all of these will contain fat which might be saturated. Sugar, jams, jellies, etc., may be eaten as desired.

Beverage. Skim milk is frequently available. You may also wish to have coffee or tea, fruit or vegetable juice, or an unsweetened beverage.

Dessert. Fruit and gelatin desserts are listed on most menus; angel food cake, sherbet, and fruit ice may be avilable. Avoid any dessert which might contain ingredients not allowed on your diet, such as fat, egg yolk, whole milk, or whipped cream.

APPENDIX F

CVRM PROGRAM CONTENT

INTRODUCTION TO THE CARDIOVASCULAR RISK CLINIC

57 A

Diseases of the cardiovascular system are responsible for one half of all the deaths in the United States each year. The major diseases of the cardiovascular system are heart attacks, strokes, and hardening of the arteries in the legs and abdominal (belly) organs. One fourth of these deaths occur in persons under the age of 65, and one male in three will experience a cardiovascular catastrophe (such as a stroke or heart attack) before the age of 60! If a person has a heart attack, there is a 50% chance of death within 5 years of another heart attack unless specific preventive therapy is instituted. The cost of cardiovascular disease is greater than 70 billion dollars per year in the United States alone. It is obvious that diseases of the heart and blood vessels are the most important health problem facing America today.

QUESTIONS

1. What causes 1/2 of all deaths in the U.S. each year?
2. Name the 3 major types of cardiovascular disease.
3. What per cent of men will experience a cardiovascular catastrophe before age 60?
4. If 100 individuals have heart attacks and are not given preventive therapy, how many will be dead after 5 years?

DEFINITIONS: HEART ATTACK, STROKE, PERIPHERAL VASCULAR DISEASE

What is a heart attack? The heart is a muscle about twice the size of your fist. Your heart beats constantly, therefore, it needs a constant supply of blood to supply the necessary food and oxygen required for function. Blood is delivered to your heart in two main arteries (large hollow tubes) called the left main coronary artery and the right main coronary artery. See figure 1. If these arteries become totally blocked with fat, cholesterol, and/or a blood clot, then part of your heart will not receive any food or oxygen. If part of your heart is deprived of food and oxygen for a sufficient period of time, then that part will die. This sudden death of part of your heart is a heart attack. See figure 2.

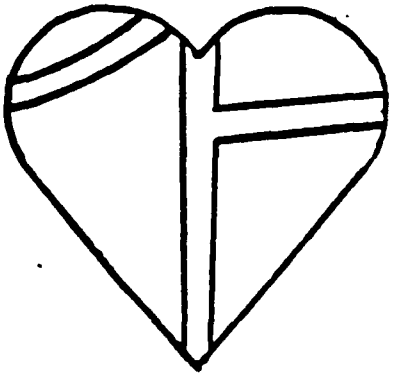


FIGURE 1

If only a small part of your heart dies as a result of a heart attack, you would probably survive. This can be compared to an eight cylinder car losing 1 or 2 cyclinders. An eight cylinder car will run on 6 or 7 cylinders. See figure number 2. If a large

part of your heart died as a result of total blockage of the left main coronary artery, then you would not survive. You would not have enough healthy heart muscle remaining to pump blood to keep the rest of your body alive. This can be compared to a 8 cyclinder car which loses six cylinders. An eight cylinder car would not run on 2 cylinders. See figure 3.

The amount of blockage as well as the location of the blockage is extremely important. If you have a massive heart attack and lose one half or more of your heart muscle, no doctor or medicine can save you.

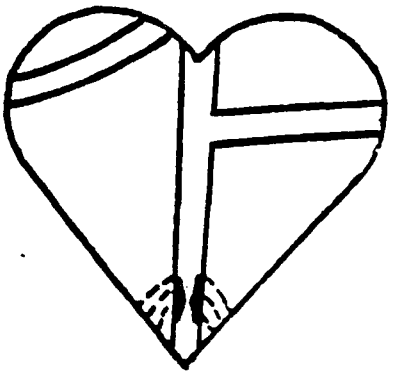


FIGURE 2

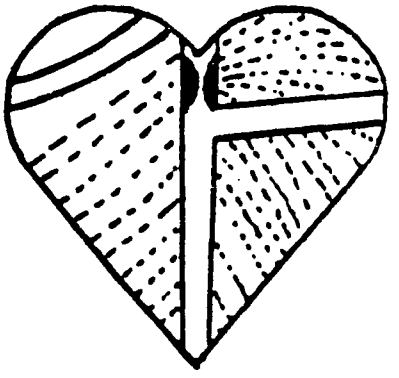


FIGURE 3

QUESTIONS

1. What is a heart attack?
2. What causes a heart attack?
3. Name the two main arteries which feed the heart.
4. What determines whether or not a person will survive a heart attack?

In a similar manner, your brain is fed by 4 large arteries. If one of these arteries became completely clogged, then part of your brain would not get enough food and oxygen. If this total blockage lasts for 5 minutes or longer, then part of your brain will die. This is a stroke. If the part of your brain which is "starved to death" is responsible for speech, then you will not be able to speak. If the part of your brain which dies is responsible for movement of your arm or leg, then you will be partially paralyzed.

If the blood vessels to your legs become obstructed, then not enough blood will be delivered to the muscle of your legs to permit normal walking. If the clogging is bad enough, then not enough blood will reach your legs to keep them alive. Gangrene will then set in.

As you can see, the main problem with cardiovascular disease is blockage of blood flow to the various parts of your body with loss of food and oxygen. When the blood flow is decreased sufficiently, parts of your body will die. This is what causes problems.

QUESTIONS

1. What is one cause of strokes?
2. What causes gangrene of the legs?
3. What is the main cause of cardiovascular disease in general?

The surgical approach to heart disease is to create new arteries which "bypass" blockages in the arteries feeding the heart. The surgeons usually remove a vein from the leg and place it between the main artery coming out of the heart (called the aorta) and the clogged arteries feeding the heart. See figure 4. These artificial arteries "bypass" the blockages and allow blood to flow to the areas of the heart which were not receiving enough blood. If a single artificial artery is created, the operation is called a "*single bypass*". If two artificial arteries are created, then the operation is called a "double bypass". A "triple bypass" is an operation in which 3 arteries are bypassed with artificial arteries.

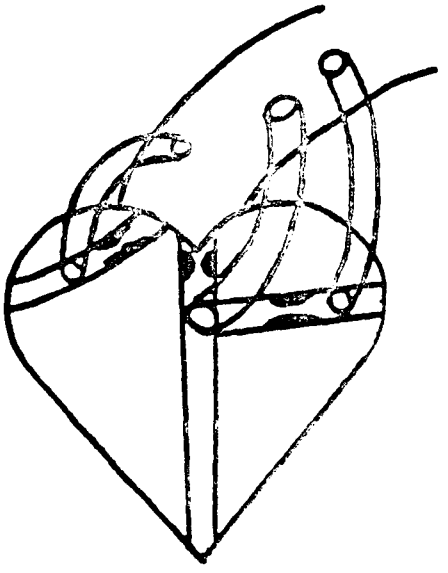
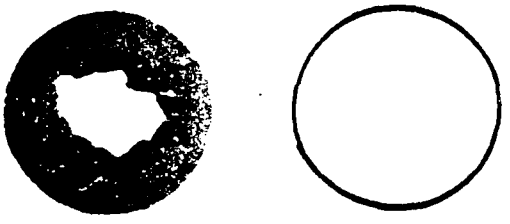


FIGURE 4

There are 5 main problems with surgery. First, the operation is very expensive (costs from $20,000.00 to $50,000.00 per operation). The second problem is that not every one gets a warning that they have heart disease. Fifteen per cent of men die suddenly from heart attacks and another 50% have nonfatal heart attacks as the first sign that the arteries to the heart are blocked. The third problem is that not every one with blocked arteries can receive surgery. The obstruction is so severe in some, that surgery is impossible.



CLOGGED ARTERY NORMAL ARTERY

The fourth problem with open heart surgery is that it doesn't last very long. Ten years after open heart sugery, one third of the artificial arteries are totally blocked and another third are almost completely obstructed by the same process which blocked the original arteries. The last problem with open heart surgery is that it is dangerous. One to five per cent of those undergoing the operation die during surgery or shortly thereafter.

It is obvious that surgery is not the best answer to the problem of heart attacks. It is better to prevent the obstruction of your arteries, than it is to "fix them" after they become obstructed. It is "easier to stay out of trouble than it is to get out of trouble".

QUESTIONS

1. How can surgeons help someone with clogged arteries to the heart?
2. What are the 5 main problems with surgery?

3. What is an alternative to surgery?

WARNING SIGNS

The main problem from cardiovascular disease is blockage of the arteries which feed the parts of your body. When the arteries to your heart are only partially obstructed (1 or 2 engines down), there is still adequate blood flow to supply its needs. However, when further clogging occurs, temporary problems can develop when your heart needs more blood than usual. The two circumstances when your heart needs more blood than usual are when you become very upset (which causes your blood pressure to rise) and when you exert yourself physically (which causes your heart rate to increase). When the needs of your heart for blood and oxygen can not be met by your arteries, an inbalance is created.

This inbalance usually causes chest pain but you may feel neck pain, jaw pain, arm pain or shoulder pain instead of chest pain. Sometimes the chest pain will move down your left arm or up to your jaw when this imbalance occurs. Shortness of breath, nausea (being sick to your stomach), and sweating often occur with the these pains.

When you stop exercising or relax after being upset, your heart rate and blood pressure fall. This decreases the demand for oxygen and food to a level which can be supplied by the arteries feeding your heart. In a short period of time, the pain will go away. These symptoms of pain with exertion or excitment, which are predictable to some extent, are the earliest signs of heart disease and are called "angina". It is very ominous when these symptoms develop while resting, since heart attacks frequently develop in those with this type of pain.

Heart pain is rarely "sharp" or "stabbing" in nature. Chest pain which changes when you cough or take a deep breath is almost never from clogged arteries to your heart.

If the arteries which feed your brain are blocked, an inbalance between the its needs and the blood supply can also occur. This temporary inbalance can cause warning signs. Some of the common warning signs for stroke are temporary loss of vision in one eye, sudden temporary loss of ability to speak or sudden temporary loss of ability to move an arm or a leg. Should these occur, you should immediately see your doctor.

If the arteries which feed your legs are blocked, a temporary inbalance between the requirement for blood of the leg muscles and the ability of the heart and blood vessels to provide blood and oxygen may occur during exercise. When not enough blood is delivered to your legs while exercising, your muscles will begin to hurt. This pain is usually relieved in a few minutes with rest. If the blockage becomes worse, you will develop pain with lighter and lighter activity. If your arteries become severely blocked, you will experience pain at rest. The next step is gangrene. This type of pain is very predictable and can be reproduced time and time again. The progression of this type of pain is usually very slow.

QUESTIONS

1. What is angina?
2. Are all heart attacks associated with chest pain?
3. Are sharp stabbing chest pains likely to be angina?
4. Heart pain frequently changes with coughing or deep breathing. T/F
5. What are the warning signs of stroke?
6. What are the signs of clogged arteries to the legs?
7. How would you know if the clogging of the arteries in the legs was getting worse?
8. Are warning signs of a stroke a medical emergency?
9. Are warning signs of a heart attack a medical emergency?

QUIET NATURE OF HEART DISEASE

Cardiovascular disease can be very "sneaky", that is, you can have blocked arteries without even knowing it. Why is this the case? The good Lord did a tremendous job when he designed your body. Your arteries are 4 times larger than necessary, to supply enough blood to the various parts of your body including the brain, heart, and legs. As a result, considerable blockage of the arteries can be present without your knowledge. Only when the last 1/4th of your artery becomes obstructed do any problems develop.

You can compare your body to an airplane, which has 4 engines but only needs one engine to fly. The plane can lose 3 engines and fly without problems, but when that last engine starts sputtering, there is nothing left to keep the plane up. In like manner, you can block 3/4th of an artery and receive no warning that anything is wrong. When that last little "bit" becomes obstructed, major problems occur.

How often have you heard a statement claiming "old John was doing just fine and then suddenly up and died of a heart attack or stroke"? In reality, "old John" was not doing "just fine". He had been clogging up his vessels (losing engines) for years. Unfortunately, he just didn't know it. It was only when his reserve blood flow was totally exhuasted, that he developed the problems which caused his death.

The pilot of an airplane would know if his plane was losing engines by looking at the instrument panel in the cockpit. We can also determine whether or not we are "losing engines". You have an instrument panel that your doctor can read. This instrument panel is called your "Cardiovascular Risk Profile". Read further and find out what this is.

QUESTIONS

1. Why can heart disease be sneaky?
2. How much reserve did you have in your system?
3. Is the statement really true that "John was doing just fine until he up and died of a heart attack"?

CAUSES OF PREMATURE CARDIOVASCULAR DISEASE

Cardiovascular diseases (heart attacks, strokes, clogg

arteries to the legs) were not always our number 1 health problem. At the turn of the century, cardiovascular diseases were only responsible for 10% of the total deaths. The rate of cardiovascular diseases increased steadily from 1900 until 1967, when they were responsible for 54% of the total deaths. The death rate from cardiovascular diseases has decreased since 1967 and they are currently responsible for 49% of all the deaths. See Figure 5

Cardiovascular diseases are more common in certain countries than in others. For example, heart attacks occur 7 times more frequently in the United States than in Japan. A large number of Japanese have moved to Hawaii, San Francisco and Los Angeles. The rate of heart attacks, in the Japanese living in Hawaii, is inbetween the low rate in Japan and the high rate in the general U.S. population. The heart attack rate, in the Japanese living in San Francisco, is higher than the rate in Hawaii. The heart attack rate, in the Japanese living in Los Angeles, is roughly the same as the rate in the general U.S. population. This clearly demonstrates that most of the difference in heart attack rates between Japan and the United States is due to different lifestyles. Genetics probably are not as important as the way we live. See Figure 6

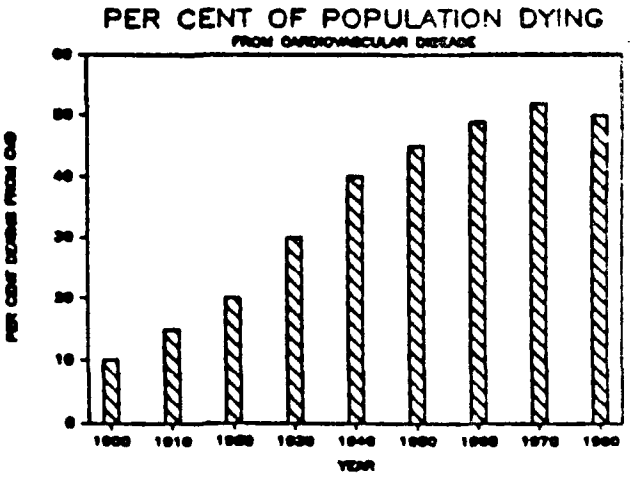


FIGURE 5

In 1948, the National Institutes of Health started a famous study of 5,209 men and women who lived in Framingham, Mass.. Multiple laboratory tests, and physical examinations were performed on these people every 2 years for many years. Heart attacks, strokes, and gangrene were followed closely. At the end of the study, the researchers were able to determine that high

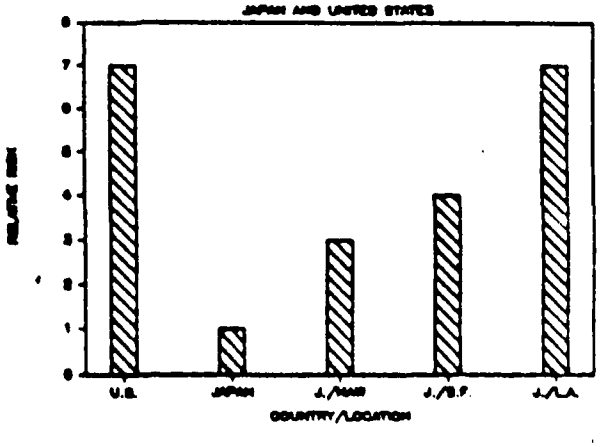


FIGURE 6

blood cholesterol, high blood pressure, high blood triglycerides, diabetes, use of tobacco, excess weight, inactive life style, low blood levels of HDL cholesterol, male sex and certain abnormalities on EKG were far more common in those who developed cardiovascular disease, than in those who did not develop problems. These were called "risk factors". See Figure 7.

It was also found that increasing blood pressure, serum cholesterol and number of cigarettes smoked per day were associated with increasing risk of developing cardiovascular disease. In addition, if one has 2 or more risk factors for cardiovascular disease, the total risk is roughly the product of the individual risk factors, not the sum. For example, if one has 3 risk factors, each of which increase the chances of having a heart attack 3 fold, the total risk would be roughly 3x3x3 or 27. The "Cardiovascular Risk Profile" is a listing of a person's risk factors and the magnitude of the increase in risk for each factor. This is the equivalent of an instrument panel in the cockpit of an airplane. If you have had several risk factors for several years, than you are likely to have "one or two engines out" or are in the process of losing your engines.

RISK FACTORS

Age	Sex
Stress	Genetics
Smoking	Diabetes
Cholesterol	High Blood Pressure
Inactivity	Triglycerides
Low HDL's	Obesity

FIGURE 7

QUESTIONS

1. Heart attacks occur times more frequently in Japan than in the U.S..
2. The heart attack rate in the Japanese living in America is identical to the rate of the Japanese living in Japan. T/F
3. What is a risk factor?
4. Name 5 risk factors.
5. What would be the increase in risk of having a heart attack in a person with 2 risk factors, each of which increases the risk by 5 fold?

THE REAL REASON DINOSAURS BECAME EXTINCT

PREVENTION

The purpose of this program is to provide information, which will help you make life style changes, which will make you less likely to develop cardiovascular disease. Until recently, the main focus in medicine has been on the therapy of a person who is sick. Treatment of cardiovascular disease is very important, but it is better to prevent it than to treat it.

Multiple studies have shown that reduction of risk factors prevents cardiovascular disease. The risk factors which can be improved are blood pressure, weight, diabetes, serum cholesterol, serum triglycerides, serum HDL cholesterol, exercise, and smoking. In the next portion of this program, you will learn a large amount of information on each one of these risk factors and what you can do to change them in a favorable manner.

CHOLESTEROL

Cholesterol is a chemical substance, which is a vital part of your body. Your body is actually made of tiny parts, called cells. Cells are so small that they can only be seen with a microscope. Each of these cells is covered by a wrapping, which is made from cholesterol and many other substances. Cholesterol is also the "backbone" of many of our body's hormones. For example, testosterone, the hormone which is responsible for the deep voice and beard in men, is made from cholesterol. Estrogen, the female hormone, and cortisone, the medicine often given in shots to help swollen and inflamed joints, are also made from cholesterol. Without cholesterol you would not exist.

If cholesterol is so vital for life, why is it associated with cardiovascular disease? As mentioned before, cardiovascular disease is caused primarily by clogged vessels. The material which blocks the arteries is made primarily of cholesterol and other fats. Cholesterol is similar to the minerals in hard water, and the pipes in a house are similar to your arteries. When hard water flows through the pipes of a house, minerals "settle out" and eventually block the pipes. The "harder" the water, the faster the minerals "settle out". In a similar manner, your pipes (arteries) can become blocked by the deposition of cholesterol in the walls of your arteries.

Water softners are commonly used to decrease the mineral content of hard water. This prevents the pipes from clogging. We can prevent our arteries from becoming blocked with cholesterol by lowering our blood cholesterol.

Elevated blood cholesterol is a major risk factor for the development of cardiovascular disease. The higher the blood cholesterol, the greater the risk. The Framingham Study showed the heart attack risk of possessing a blood cholesterol of 335 to be four times higher than the risk of having a blood cholesterol of 185! See Figure 8. In the United States, the upper limit of normal for blood cholesterol is 295 points. The average blood cholesterol of a 50 year old white American male is 245. In Japan, the upper limit of normal for blood cholesterol is 195 points and the average blood cholesterol of a 50 year old Japanese male is

195 points and the average blood cholesterol of a 50 year old Japanese male is only 160.

This is one of the main reasons why heart attacks are seven times more common in Americans than in the Japanese. When the Japanese move to the United States and adopt our "western" lifestyles and diet, their cholesterol levels increase. This is followed by a dramatic increase in the risk of developing heart attacks.

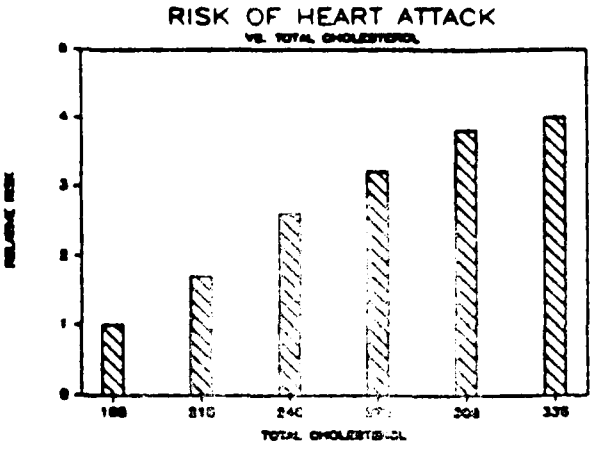


FIGURE 8

There are many factors which affect your blood cholesterol level. See Figure 9. Diets which are high in cholesterol, total fat and saturated fat increase blood cholesterol. Polyunsaturated fats, on the other hand, will lower your blood cholesterol. It is usually easy to tell if a fat or oil is saturated or polyunsaturated. Saturated fats will harden when placed in a refrigerator, but polyunsaturated fats will remain liquid in the refrigerator. The only exception is palm oil, which will remain liquid in the refrigerator but contains large amounts of saturated fat.

FACTORS AFFECTING CHOLESTEROL

Dietary Cholesterol	Age
Polyunsaturated Fats	Sugar
Medications	Exercise
Saturated Fats	Weight
Coffee	Fiber

FIGURE 9

If you gain weight your blood cholesterol will rise. Weight loss will lower your blood cholesterol. Diets which contain large amounts of sugar can raise your blood cholesterol but diets which are high in fiber can lower it. The best diet for your heart is one which is low in cholesterol, total fat, saturated fat, and sugar, while containing high amounts of fiber and polyunsaturated fats. Your diet should only contain enough calories to maintain ideal body weight.

Cholesterol levels increase with age in many, even if diet and weight are held constant. Medications can also affect your cholesterol. Some common blood pressure medications can raise your blood cholesterol, while others can lower it. Exercise can lower your blood cholesterol. Alcohol, in excessive amounts, can raise your cholesterol level.

Women have lower serum cholesterol levels than men in general, and there is great individual variability. Two percent of the population will have very high blood cholesterol levels, no matter what they eat. Another two percent of the population will have very low levels, despite consumption of the worst possible diet. For 96% of the population, diet is very important in determining blood cholesterol level and risk of cardiovascular disease.

As you can see, there are many things which affect your blood cholesterol level. It is not surprising, that there is

considerable confusion and debate on the effect of diet on blood cholesterol level.

QUESTIONS

1. Cholesterol can be compared to what in hard water?
2. How is cholesterol involved in cardiovascular disease?
3. What is the increase in risk if an individuals has a blood cholesterol of 335 compared with 185?
4. Name the 6 features of the ideal cardiac diet.
5. Name 4 non-dietary factors that influence blood cholesterol level.
6. What is the average blood cholesterol in a 50 year old white American male?
7. What is the average blood cholesterol in a 50 year old Japanese male?

The average American diet contains 500 mg of cholesterol per day and 40% of the calories come from fats. Saturated fat (the type of fat) is responsible for 20% of the calories in the diet and polyunsaturated fat (the good fat) is responsible for less than 5%. In Japan, less than 10% of the total calories come from fat and only 3% of the calories come from saturated fat. In some areas of Japan, only 100 mg of cholesterol are consumed per day. In essence, we eat 5 times as much cholesterol per day and consume almost 7 times as much saturated fat as the Japanese. This is the reason blood cholesterol levels are so much lower in Japan than in the United States and why the heart attack rate is so much lower. See Figure 10.

UNITED STATES vs JAPANESE DIET
FAT & CHOLESTEROL CONTENT

	Cholesterol/Day	Saturated Fat (% Total Calories)
U.S. DIET	500 mg	20%
JAPANESE DIET	100 mg	3%

FIGURE 10

Many researchers believe the best diet is one in which the total daily cholesterol is limited to 100 mg and total fat is limited to 20% or less of the total calories. Saturated fats should comprise less than 5% of total calories. Current recommendations from the National Institutes of Health and the American Heart Association, for the general population, are that total cholesterol be limited to 250-300 mg per day and fat be restricted to 30% of total calories. If a person has an elevated blood cholesterol or established cardiovascular disease, the total cholesterol should be limited to 150-250 mg per day and total fat be reduced to 25% of the total calories. Saturated fats should comprise less than 10% of the total calories.

QUESTIONS

1. The average American diet contain how many mg of cholesterol per day?
2. What is the ideal cholesterol consumption per day?
3. What is the average consumption of saturated fat in the United States?
4. What is the average saturated fat consumption in Japan?

CHOLESTEROL AND SATURATED FAT CONTENT OF COMMON FOODS

Foods which are low in cholesterol, low in saturated fat, and relatively high in polyunsaturated fat are very desirable for the heart. Vegetables, oats, fruits, whole grain cereals, and beans have almost no cholesterol and are very low in fat. The small amount of fat in vegetables and beans is usually polyunsaturated. Beans and oats are particularly high in fiber. The fiber in beans binds to cholesterol in the intestines, causing cholesterol elimination in the stool. By consuming a diet high in beans and oats, you will not only decreased the amount of cholesterol and fat in your diet, but will also increase cholesterol loss in your stool. The child's rhyme "beans, beans, good for the heart, the more you eat the more you . . . . ., the more you . . . . the better you feel, so lets eat beans for every meal" is scientifically very accurate.

Why does the medical profession encourage the use of fish, chicken, low fat cottage cheese, yogurt, and eggs substitutes for beef, pork, cheese, whole eggs and whole milk? Tables 1 and 2 list the cholesterol and fat content of common foods. In the section on meats, the foods with the least cholesterol are white fish, clams, scallops, oysters, and water packed tuna. There is very little difference in the cholesterol content of chicken, lean veal, beef, pork and lamb. Organ meats (such as liver, brains etc) and whole eggs are extremely high in cholesterol.

If chicken, beef, and pork have similar amounts of cholesterol, why are chicken and fish recommended as substitutes for cheese, beef and pork? The answer lies in the columns marked "total fat" and "saturated fat". Chicken, without skin, has 1/3 the saturated fat of the leanest veal, beef, and lamb. Chicken, without skin, has 1/10 of the saturated fat contained in common hamburger and the finest steaks (T-bone etc). Since the total amount of saturated fat in the diet, is an important factor in the blood level of cholesterol and risk of heart attacks, the low fat content of chicken without skin offers a distinct advantage over beef and pork. The chicken, to be beneficial, must be cooked and consumed without its skin. The total and saturated fat content of most chicken fried with the skin, is almost identical to the total and saturated fat content of lean hamburger. Changing from hamburger to fried chicken does not lower blood cholesterol. It is similar to changing from "Exxon Oil" to "Texaco Oil".

The saturated fat content of white fish, clams, and oysters is 6 times lower than chicken, without skin, and 65 times lower than common hamburger. The low cholesterol and saturated fat content of fish make it an ideal part of a "good heart" diet. Fish can be ruined, if it is breaded and deep-fat fried in a highly saturated oil.

Eggs are very high in cholesterol (500 mg for 2 medium sized

TABLE 1

CHOLESTEROL AND FAT CONTENT OF COMMON FOODS

FOOD	CHOLESTEROL (mg/3.5 oz)	TOTAL FAT (gm/3.5 oz)	SATURATED FAT (gm/3.5 oz)
FISH			
white fish, clams, scallops, oysters, water-packed tuna	66	0.9	0.2
shrimp, crab, lobster	112	1.7	0.2
salmon	75	6.9	1.7
POULTRY			
chicked and turkey, no skin	87	4.9	1.3
duck and goose, with skin	91	33.4	8.0
VEAL			
10% fat-trimmed roasts, veal cutlets, and chops	99	11.1	4.7
15% fat-untrimmed roasts, chops, and cutlets	99	16.9	7.1
BEEF, PORK, AND LAMB			
10% fat-ground sirloin, trimmed lean beef, and lamb	90	10.0	3.7
20% fat-ground chuck, untrimmed beef and lamb roasts	90	19.1	8.3
30% fat-ground beef and pork, well-marbled steaks, chops (T-bones, etc.), and ham	90	30.7	12.9
ORGAN MEATS	300-2,000	4.8	1.6

Adapted from Table 6, Symposium on Lipid Disorders, MEDICAL CLINICS OF NORTH AMERICA, 66(2), March 1982. William E. and Sonja L. Conner.

TABLE 2

CHOLESTEROL AND FAT CONTENT OF COMMON FOODS

FOOD	CHOLESTEROL (mg/3.5 oz)	TOTAL FAT (gm/3.5 oz)	SATURATED FAT (gm/3.5 oz)
EGGS			
white	0	0	0
egg substitutes	0	4.2	1.2
whole	504	11.5	3.4
VISIBLE FATS			
Most vegetable oils	0	100	13
soft veg. margarines	0	81	16
soft shortenings	0	100	25.8
butter	227	81	49.8
coconut oil, palm oil	0	100	74.6
cocoa butter (chocolate)		100	74.6
CHEESES			
dry curd cottage cheese, tofu, pot cheese, low-fat cottage cheese	6	2.1	0.9
cottage cheese, part skim ricotta	29	7.5	4.6
low fat cheddar, part skim mararella, skim American	58	18.2	10.2
cheddar, roquefort, swiss, brie, jack, American, cream cheese, velveeta, cheese spreads	106	35.0	20.6
FROZEN DESSERTS			
water ices	0	0	0
sherbert or frozen yogurt	4	1.2	0.8
ice milk	14	5.1	3.2
ice cream	40	10.6	6.6
MILK			
skim milk	2	.1	(less than) 0.1
butter milk	4	1.0	0.6
2% milk	10	2.0	1.1
whole milk (3.5% fat)	18	3.5	2.1
nondairy creamers	0	11.0	8.5

Adapted from Table 6, Symposium on Lipid Disorders, MEDICAL CLINICS OF NORTH AMERICA, 66(2), March 1982. William E. and Sonja L. Conner

eggs). A single egg yolk has roughly 300 mg of cholesterol. This is equal to the maximum daily amount recommended by the NIH, for a normal individual, and is twice the amount which should be eaten by a person with known heart disease and/or high blood cholesterol. One preventive cardiologist refers to egg yolks as "death balls". Egg whites, however, have no chole terol or fat and are a good substitute for whole eggs in receipes. Two egg whites can be substituted for one whole egg in receipes. The high quality protein of egg whites and relative low cost make this substitution feasible. The egg yolks should be discarded rather than eaten.

Other less desirable foods are chocolate, palm oil, coconut oil, regular cheese, most "low-fat" cheeses, butter, whole milk, stick margarine, organ meats (such as liver, brains etc.) and ice cream. One should substitute soft margarine, polyunsaturated vegetable oils, low-fat cottage cheese, skim or buttermilk, and yogurt for these items in the diet. Skim and buttermilk are much lower in fat and cholesterol than "low-fat" or whole milk. Low-fat (2%) milk has 5 times as much cholesterol, 12 times as much total fat, and 15 times as much saturated fat as skim milk. Buttermilk has 1/2 the cholesterol, total fat, and saturated fat of low-fat (2%) milk. Low-fat milk has less fat than whole milk, but is really not low in fat. Skim and butter milk are much better. See Figure 11. The greatest reduction in blood cholesterol occurs when the total fat content of the diet is reduced. For this reason, frying should be avoided as well and coupled with a reduction in the use of all visible fats, such as soft margarine and vegetable shortenings.

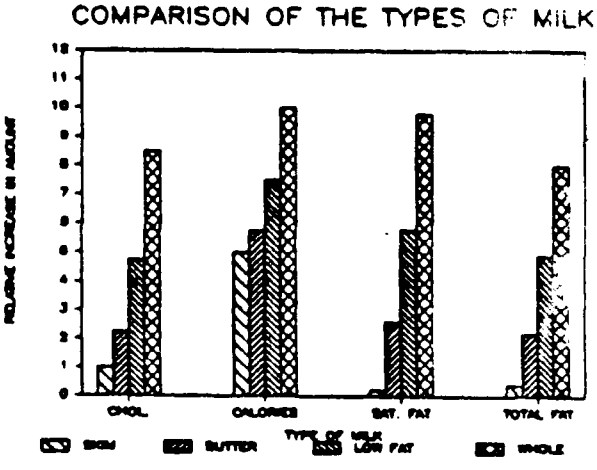


FIGURE 11

Some interesting examples will illustrate the benefits of ingesting fish and chicken without skin as opposed to beef, pork, lamb and cheese. In a 3 ounce hamburger, there are 12.9 gms of saturated fat (see Table 2). In 3 ounces of white fish, there is only 1/5th of a gm of saturated fat. The saturated fat content of hamburger is 65 times higher than fish. You can eat 3 x 65 or 195 ounces of fish before obtaining the same amount of saturated fat in 1 small hamburger. This is over 12 lbs! If you snacked on 3 ounces of cheddar cheese, you would eat 20.6 gms of saturated fat. The saturated fat content of cheese is 100 times higher than the saturated fat content of fish. To consume the same amount of saturated fat, you would have to eat 100 x 3 or 300 ounces of fish. This is over 18 lbs!

The advantages of fish compared to beef, pork, and cheese are obvious. The Japanese eat large quantities of fish and very little beef, pork, and cheese. This is reason their blood cholesterol levels are so much lower than ours and why their heart attack rate is also much lower.

Butter is the only visible fat which contains cholesterol. Fifty per cent of its calories come from saturated fat. Soft margarine or vegetable oils should be substituted for butter in cooking or in general use. Most vegetable oils are good with low quantities of saturated fat and relatively high quantities of polyunsaturated fat, however, reduction in total fat content of the diet produces the greatest reduction in blood cholesterol. Substitute soft margarine for butter whenever possible, but limit its use overall. Palm oil and coconut oil are 2 vegetable oils, which contain large amounts of saturated fat and should be avoided.

A standard American breakfast is 2 eggs, sausage, bacon or ham, and toast with butter. This meal contains very high amounts of cholesterol and saturated fat. A standard American lunch is a cheeseburger, french fries (typically fried in a highly saturated fat) and a milk shake. This is a large part of the reason why the heart disease rate in the United States is so high! The old saying "eat a good breakfast, best meal of the day" was probably invented by an undertaker.

QUESTIONS

1. What are the main advantages of beans, oats, and vegetables?
2. What dairy products are particularly high in cholesterol and saturated fats?
3. Why should chicken and fish be substituted for beef and pork?
4. How many pounds of white fish would one have to eat to obtain the same amount of saturated fat contained 1 cheese burger? (1 cheeseburger contains 3 ounces of hamburger and 1 ounce of American cheese)
5. How many pounds of chicken would you have to eat to obtain the same amount of saturated fat contained in a 12 ounce T-bone steak?
6. What 2 vegetable oils have large amounts of saturated fat?

HIGH DENSITY LIPOPROTEIN CHOLESTEROL (HDL)

Clogging of your arteries occurrs because the cholesterol in your blood "settles out" slowly throughout your lifetime. Fortunately, your body has a cholesterol removal system which can reverse this process. The name of this removal system is the HDL system. HDL stands for High Density Lipoprotein. The cholesterol which is being removed from the walls of the arteries can be measured. It is called HDL cholesterol and is commonly referred to as the "good cholesterol".

Arteries become "plugged up" by cholesterol because more cholesterol is put into the walls of the arteries, than is removed from them. The higher the total blood cholesterol, the faster it is deposited into the walls of the arteries. The higher the HDL cholesterol, the faster the cholesterol removal. The higher the HDL cholesterol level, the lower the risk of heart attack. An individual with an HDL cholesterol of 25 is 7 times more likely to have a heart attack, than an individual with an HDL cholesterol of 85. See Figure 12.

If you have a very high total blood cholesterol and a low HDL cholesterol, then clogging of your arteries proceeds very quickly. If you have low total cholesterol and a very high HDL cholesterol, then your arteries will probably stay open. The lower the ratio of

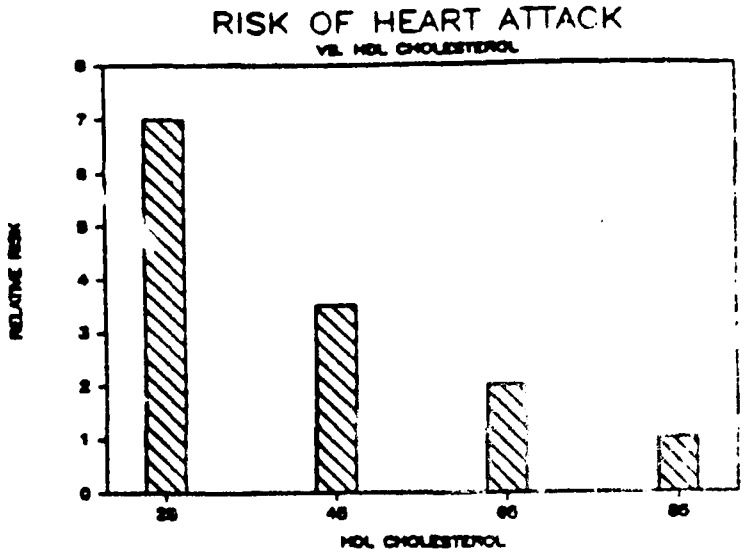


FIGURE 12

total cholesterol to HDL cholesterol the better. Ratios which are less than 4.0 are usually safe. Ratios which are higher than 4.5 are associated with an increasing risk of coronary artery disease. Ratios as high as 8 are very risky. The ratio of total cholesterol to HDL cholesterol is the best predictor for heart attacks.

Individuals, who live to be 90 or older, generally have very high levels of HDL cholesterol and low ratios of total cholesterol to HDL cholesterol. Your removal system can be compared to "Pac-man", the popular video game ("Pac-man" is a registered trademark of the Bally Manufacturing Company, Chicago Illinois). The "Pac-man" "gobbles up" everything in its way. In a like manner, your HDL system "gobbles up" the cholesterol which has been deposited in the walls of your arteries. It is your internal vacume cleaning system!

There are several things which can increase your HDL cholesterol, "Pac-man", and reduce your risk of heart attack. The most important are aerobic exercise, moderate alcohol consumption, weight loss, reducing sugar in your diet, estrogen (for women who have gone through the "change of life"), low cholesterol and low saturated fat diets, ingestion of polyunsaturated fats, and certain medications (such as nicotinic acid). There is also evidence that the fiber in beans can raise HDL cholesterol levels in some individuals.

There are several things which lower your HDL cholesterol. The most important are cigarette smoking, inactivity, weight gain, diabetes mellitus, excessive alcohol intake (it poisons the liver which makes the HDL), high amounts of sugar consumption in your diet, testosterone (the male hormone) and certain blood pressure medications. There is also evidence that diets high in saturated fats can lower your HDL cholesterol.

QUESTIONS

1. What is HDL cholesterol?
2. What determines whether or not an individuals's arteries will become "clogged up" with cholesterol?
3. People who live to be 90 or older have low levels of HDL cholesterol. T/F
4. What can HDL be compared to?
5. Name 6 things that can raise HDL cholesterol levels.
6. Name 5 things that can lower HDL levels.

BENEFITS OF CHOLESTEROL REDUCTION

Many studies have shown that cholesterol reduction with diet or medications, reduces the risk of heart attack. See Figure 13.

FIGURE 13 BENEFITS OF LOWERING CHOLESTEROL

DIET

1. Finnish Mental Hospital Study . . . . Heart Attacks Cut in Half
2. OSLO Study . . . . . Heart Attacks Cut in Half
3. Los Angeles V.A. Study . . . . . Heart Attacks Cut in Half

DRUGS

1. Cholestyramine . . . . . Heart Attacks Reduced 25-50%
2. Nicotinic Acid . . . . . Heart Attacks Reduced 27-44%
3. Clofibrate . . . . . Heart Attacks Reduced 20-62%

The first study, which showed the benefits of a low cholesterol and low fat diet, was done in Finland. The patients in 1 large mental hospital were fed a low cholesterol, low saturated fat, and high polyunsaturated fat diet. The patients, in a different mental hospital, were fed the standard Finnish diet, which contains high amounts of cholesterol, saturated fat, and small amounts of polyunsaturated fat. At the end of 6 months, the blood cholesterol levels in the group on the low cholesterol and low saturated fat diet were 60 points lower than the cholesterol levels in the group on the regular diet. After 6 years of observation, there were 2 times as many heart attacks and deaths due to heart attacks in the patients who were given the regular diet when compared with those given the cholesterol lowering diet.

The diets were switched, with the patients in the first hospital receiving the standard Finnish diet and the patients in the second hospital receiving the low cholesteol, low saturated fat and high polyunsaturated fat diet. After 6 months, the cholesterol levels of the patients in the first hospital increased significantly, while the cholesterol levels in the patients in the second hospital fell markedly. After 6 years of observation, there were twice as many heart attacks and deaths due to heart attacks, in those consuming the standard Finnish diet when compared to those on the cholesterol lowering diet. This proved that the diet was responsible for the decrease in heart attacks.

This same study was performed in Los Angeles at a Veterans Administration Domicillary and in Oslo Norway. In each of these studies, the same thing was found. Reduction of blood cholesterol by diet was capable of decreasing the number of heart attacks and death due to heart attack by a factor of 2.

In March 1984, Time magazine published the results of a very famous study which showed that cholesterol lowering with medications decreased the risk of heart attacks and deaths due to heart attacks. In essence, patients were given a medication (Cholestyramine) which lowered blood cholesterol (by 45 points on the average) or a fake medicine (which did not lower blood cholesterol). The patients given the medication, which lowered blood cholesterol, had 25-50% fewer heart attacks and deaths due to heart attacks, than the patients given the fake medicine. Three

other studies have show similar results with other cholesterol lowering medications, such as, nicotinic acid and clofibrate.

The most exciting development in preventive cardiology has been the demonstration that a person can "clean out" his own arteries, if the blood cholesterol is lowered sufficiently and the HDL cholesterol is raised enough. This was demonstrated in a 39 year old woman, who had severe clogging of the arteries to her heart and chest pain so severe, tha she was barely able to walk up a single flight of stairs. After 2 years of therapy with a cholesterol lowering diet and medications, which reduced her cholesterol from well over 300 to less than 200, she was totally free of chest pain. A repeat study of her heart showed almost complete removal of all blockages. Similar results have been seen in many other individuals when they adopted a "healthy heart" life style.

QUESTIONS

1. A cholesterol lowering diet has been shown to reduce heart attacks and deaths due to heart attacks by a fator of .
2. How many studies have shown that diet can decrease the number of heart attacks?
3. Cholesterol lowering medications do not prevent heart attacks. T/F

WEIGHT AND CALORIC DENSITY

Ideal body weight (IBW) is the weight which is associated with the lowest mortaltiy for age and height. The IBW has been determined by studying death rates by weight of people who obtained life insurance. The risk of death increases with increasing body weight above the IBW. See Figure 14.

The average American weighs 20% more than the IBW and 80% of the population would benefit by weight reduction. Weighing 20% more than the IBW, increases the likelihood of dying from a heart attack by 25%. Weighing 40% more than IBW, increases the chances of dying from a heart attack 2 fold. Weighing 100% or more above IBW (morbid obesity), increases the risk of dying 12 fold. One fourth of the morbidly obese die every 10 years, beginning at age 20. Hence, it is very uncommon for morbidly obese individuals to live past age 60.

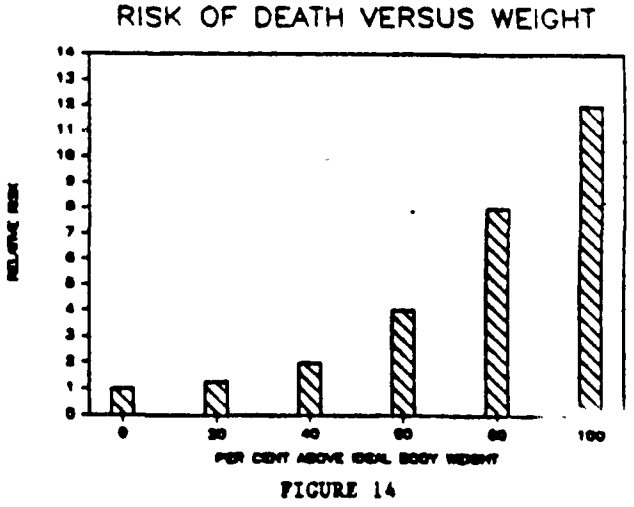


FIGURE 14

Weight gain can adversely affect health. These adverse effects include high blood pressure, elevation in blood

cholesterol, reduction in HDL cholesterol ("Pac-man"), increase in blood sugar (diabetes) and harmful social stigmata. If a lean individual and an obese individual apply for the same job with equal qualifications, the job will almost always be given to the leaner individual. Hence, there are many health relatved and social reasons for maintaining ideal body weight.

Every person has a "thermostat" for food and weight. This "thermostat" is located in the brain. If you gain weight above your "set weight", then weight loss will be spontaneous. If you lose weight (with a diet etc.), then you will feel very hungry until you regain your weight. This is why it is so difficult to maintain weight loss. This "set weight" is determined by the end of puberty. If a child is overweight at the end of puberty, there is only 1 chance in 30 that the child will ever maintain ideal body weight. The best way to deal with excess body weight is to prevent it in children.

Your "thermostat" can be set at a lower point by regular exercise, and by altered eating patterns. The most important aspect of a weight reducing diet is eating foods which have low caloric density. Caloric density refers to the number of calories in an ounce. Eating is a very social event and most don't like to eat only a few bites of food at a meal. Tasting, chewing, and swallowing are very enjoyable. Furthermore, most individuals eat until their stomach is slightly distended.

Eating foods which do not have very many calories per bite, enables you to take more bites, taste more food, and have a certain amount of stomach stretch without eating too many calories. The foods which are calorically dense (have many calories per bite) are beef, pork, oils, cheese, butter, margarine, nuts, peanut butter and foods which contain refined sugars such as ice cream, puddings, pies, cakes, candies etc. Foods which are not calorically dense are vegetables, beans, fruits, fish, and chicken (without skin and not fried).

To gain an appreciation for caloric density consider the following examples. See Table 3. Three ounces of cheddar cheese contains 400 calories. Three ounces of asparagus contains only 26 calories. If you were on a diet and snacked on only 3 ounces of cheese, you would ingest a large amount of calories in only a few bites. If you snacked on asparagus, you could eat 3 lbs before eating the same number of calories. You probably would not be able to "fit" 3 pounds of asparagus in your stomach or afford it. Hence, you would eat fewer calories and lose weight. Other striking examples show that you can eat 10 lbs of chinese vegetables before ingesting the same number of calories contained in 1 small piece of pecan pie.

The most calorically dense food items are oils and fats. Three ounces of lard or corn oil contains over 900 calories. For this reason, fried foods should be avoided in individuals who desire to lose weight. Table 3 contains information on the number of calories in 3 ounces of common foods. Note the difference between the various meats, dairy products, fruits and vegetables.

A popular form of dieting is to avoid the good tasting foods with high caloric density monday thru friday. "Normal" eating patterns are followed on the week ends. That is, do not eat beef, pork, cheese, fried foods, and sweets monday thru friday, but eat as you like on week ends. Vegetarian snacks are encouraged. At

Laughlin AFB, this pattern of eating resulted in an average weight loss of 15-20 pounds. Several individuals lost up to 70 pounds in one year. This form of dieting will also limit the amount of cholesterol and saturated fat in the diet. This will also lower cardiovascular risk.

TABLE 3

FOOD	CAL./3.5 Oz.	FOOD	CAL./3.5 Oz.
MEATS		FISH	
hamburger	327	sea bass	96
ham	371	tuna in water	127
white chicken w/o skin	173	tuna in oil	311
dark chicken w/o skin	205	shrimp (boiled)	91
white chicken w. skin	222	shrimp (fried)	381
bacon	692	filet of sole	68
DAIRY PRODUCTS		DAIRY PRODUCTS	
cheddar cheese	400	skim milk	35
swiss cheese	360	low fat (1%) milk	41
cream cheese	350	whole milk	61
plain no fat yogurt	39	butter milk	41
plain low fat yogurt	54	low fat cottage cheese	75
ice cream	250	cottage cheese	100
VEGETABLES		FRUITS	
green beans	38	orange	40
dried beans cooked	10-150	banana	85
potato	76	cantaloupe	30
tomato	22	apple	57
asparagus	26	strawberries	30
rice cooked	110	peach	42
DESSERTS		NUTS OILS	
ice cream	250	corn oil	900
sherbet	140	lard	900
pies	250-350	pecans	696
milk choc. nuts	542	raw peanuts	543
chocolate fudge	390	pea nut butter	576
no-fat yogurt sweetened with equal	40	cashew nuts roasted	560

QUESTIONS

1. How many pounds of green beans can you eat before ingesting the same number of calories contained in 5 ounces of pecans?
2. How much artifically sweetened no-fat yogurt can you eat before ingesting the same number of calories contained in 8 ounces of ice cream?
3. How many tomatoes can you eat before ingesting the same number

of calories contained in a 3.5 ounce hamburger?
4. How much filet of sole can you eat before ingesting the same number of calories contained in a 3.5 ounce portion of ham?
5. What is ideal body weight?
6. What is the increase in risk of cardiovascular disease when one weighs 100% above ideal body weight?
7. What are the harmful effect of weight gain?
8. What are 2 mechanisms which help maintain weight loss?
9. When is the thermostat for weight set?
10. What is the likelihood that an obese adolescent will ever be thin?

SMOKING

Smoking is a major risk factor for the development of cardiovascular disease. In the early 60's, the Surgeon General published a famous medical study which proved that smoking caused lung cancer and was associated with an increased risk of cancer of the mouth, throat and the tube which connects your mouth to you stomach. The study demonstrated a 40 fold increase in the risk of dying from lung cancer in smokers compared to non-smokers. See Figure 15.

In actuality, only 1 smoker in 20 will die from lung cancer, hence, the threat of lung cancer is not great to a person who really enjoys smoking (19 times out of twenty he will not die of lung cancer).

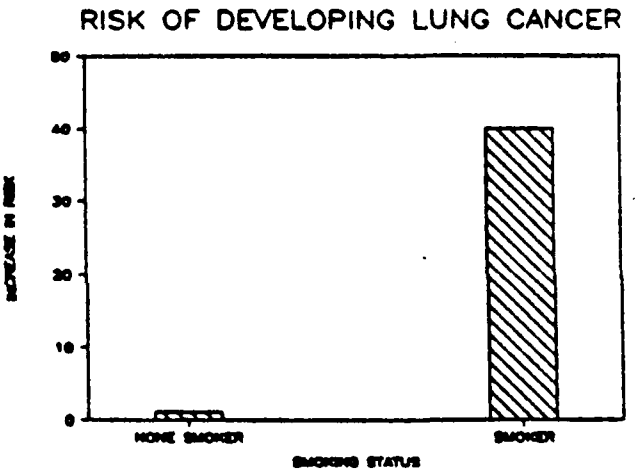


FIGURE 15

The Framingham Study and many other studies have demonstrated that smoking increases the risk of cardiovascular disease especially, the risk of heart attack. The studies have also showed that the risk from smoking increases with increasing number of cigarettes smoked per day. For instance, if you smoke 1/2 a pack of cigarettes per day, you will be 3 times more likely to die of a heart attack than a non-smoker. Smoking 1 pack of cigarettes per day will increase your risk of heart attack by 5 fold and 2 packs per day increases your risk of dying from a heart attack 8 fold. See Figure 16. Smoking cigars or a pipe increases your risk of a heart attack 2 fold. Since cardiovascular diseases are responsible for one half of all deaths in the country, this huge increase in risk of death is a much stronger reason why you should stop, if you currently smoke.

Smoking produces this huge increase in heart attacks three ways. Smoking lowers your blood HDL cholesterol, the "Pac-man". The more cigarettes you smoke, the greater the reduction in HDL cholesterol. Smoking also increases the tendency for your blood to clot. If your arteries are partially blocked, the final step in

the development of a heart attack is usually the formation of a blood clot. A blood clot will totally stop blood from getting to part of your heart and cause a heart attack. By increasing the tendency for blood to clot, smoking increases your risk of heart attack. Finally, every artery in the heart is surrounded by thin layer of muscle. Muscle has the ability to contract and become smaller. The poisons in tobacco cause the muscles surrounding the arteries, which feed your heart, to contract soon after you take the first puff. This causes a reduction in the size of the arteries. See Figure 17. If the artery is already slightly clogged from cholesterol, the additional blockage caused by this "spasm" of the artery can totally shut off the blood supply to your heart and cause a heart attack, possibly death.

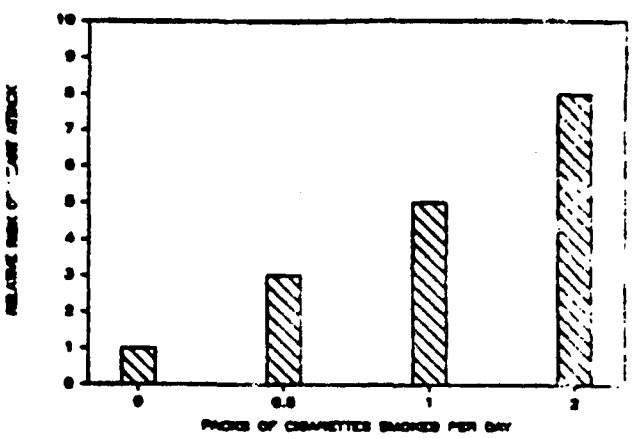
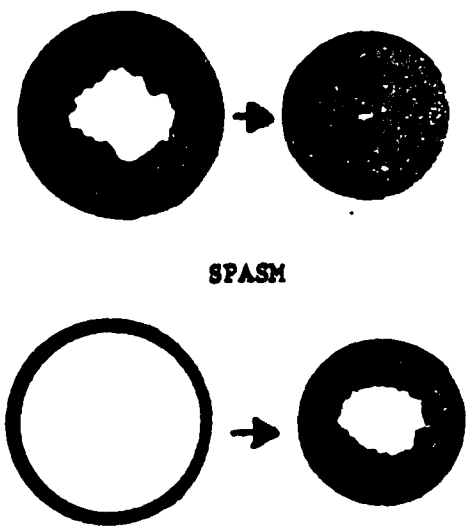


FIGURE 16

Smoking is especially risky in women over 35 years of age, who take birth control pills. In this group, the increase in risk of heart attack can be as high as 250! For this reason, many gynecologists will not give birth control pills to women over age 35 who smoke.



SPASM

FIGURE 17

Many smokers joke that "stopping smoking doesn't allow you to live longer, it just makes life seems longer". In actuality, the day you stop smoking, your risk of death from all causes is cut in half. If you stop smoking before the age of 35 the risk of dying is reduced 3 fold. Smoking a pipe or cigar increases your risk of death from all cardiovascular disease by 2 fold. Chewing tobacco and snuff have also been shown to dangerous to your health. Smoking cigarettes which are "low in tar and nicotine" will not decrease the risk of cardiovascular disease. The number of cigarettes used per day is what determines risk.

Breathing the smoke of others is also dangerous. Wives of husbands who smoke are twice as likely to die of lung cancer, compared to wives of husbands who do not smoke. Being around

people who smoke, produces the same effect as smoking 3 cigarettes per day (10 cigarettes per day increases the risk of heart attack by 3 fold). Individuals with angina, who are exposed to someone else's cigarette smoke, develop chest pain much more easily than they would if they were not exposed to smoke. Children, born to mothers who smoke, are 2-17 times more likely to have birth defects than children born to mothers who do not smoke.

Additional research has demonstrated that smoking is also associated with cancer of the stomach, bladder, pancreas, and kidney. Cancer is responsible for 17% of all the deaths in the country. Smoking is associated with one half of all cancer deaths. Emphysema is responsible for 10% of all of the deaths in this country. Emphysema is almost exclusively caused by smoking. Putting it all together, smoking will cause the death of the smoker 7 times out of 10 (not the 1 chance in twenty from lung cancer)!

Fortunately, there is more help now, if you desire to quit, than ever before. There are 2 medications which can help you quit. Ask your doctor for help if you can't quit by yourself. It is important to remember that the average smoker "quits" 7 times before quitting for good. Don't "quit being a quitter".

QUESTIONS

1. Smoking is associated with a fold increase in the risk of developing cancer of the lung.
2. Smoking 1/2 pack per day increases the risk of dying from a heart attack fold.
3. Smoking 1 pack per day increase the risk of heart attack fold.
4. Smoking 2 packs per day increases the risk of heart attack fold.
5. Pipe or cigar smoking is safe. T/F
6. Stopping smoking reduces the risk of dying fold.
7. Smoking increases the risk of heart attack by 3 mechanisms, name them.
8. Smoking will kill the person that smokes times out of ten.
9. Smoking is safe for pregnant women. T/F
10. Being around someone who smokes is irritating but not harmful to one's health. T/F

HYPERTENSION

Hypertension or high blood pressure is a major risk factor for the development of premature cardiovascular disease. The risk of cardiovascular disease increases with increasing systolic blood pressure (the top number) and with increasing diastolic blood pressure (the bottom number). The safest blood pressure is

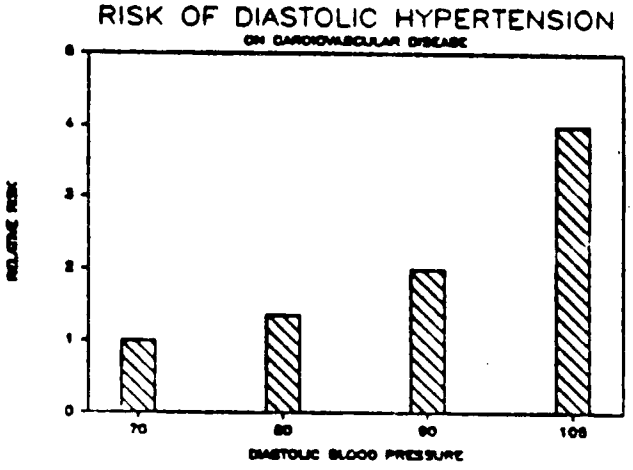


FIGURE 17A

105/70 or lower. As blood pressure increases above these levels (top or bottom), the risk of cardiovascular disease increases. See Figures 17A and 18. The increase in risk, if your systolic blood pressure (the top number) is 135 rather than 105, is 2 fold. There is a 35% increase in risk, if the diastolic blood pressure is 80 rather than 70. Physicians, however, will only treat you for high blood pressure with medications when your blood pressure is over 145 on the top and 90-95 on the bottom. Yet, it is much safer to have a much lower blood pressure. Why don't physicians treat those whose blood pressure is above 105/70 and below 145/90-95 with medications?

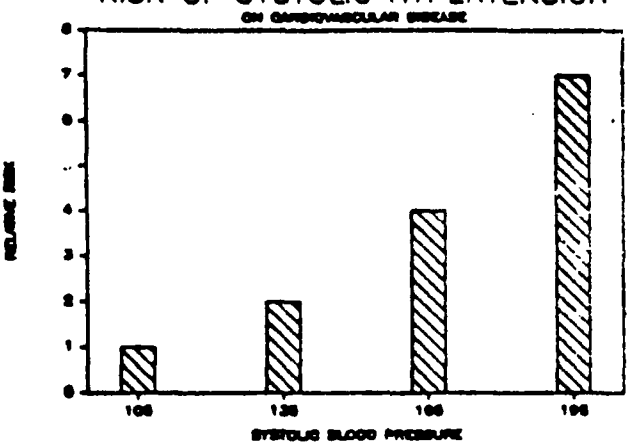


FIGURE 18

Reduction of blood pressure with medications has been a tremendous benefit to our society. Treatment with drugs, produces a 13 fold reduction in cardiovascular catastrophies in those whose diastolic blood pressures (bottom number) are above 115. If the diastolic blood pressure is between 105 and 115, treatment with medications will lower cardiovascular catastrophies by 3 fold. If the diastolic blood pressure is between 90 and 105, it is not clear that further reduction of the pressure with medication helps. One large study showed a reduction of cardiovascular catastrophies of only 20%, when medications were used to lower diastolic blood pressures between 90 and 105 (although the risk of having a heart attack or stroke is 2 to 4 times higher than if the diastolic blood pressure was less than 70). Two other studies showed no improvement when treating diastolic blood pressures between 90 and 105 with medications. An additional study showed that reducing diastolic blood pressures between 90 and 105 with medications increased the risk of cardiovascular disease.

If your blood pressure is 140/90, you are roughly 2 times more likely to have a heart attack or stroke than if your pressure is 105/70. The reason that medications do not reduce the risk of heart attack, with these "low levels of high blood pressure", is that the side effects of the blood pressure medications are more harmful than the benefit of the reduction in blood pressure. In other words, the "cure is worse than the disease" for "low levels" of hypertension. Some of the bad side effects of blood pressure medications are elevated blood cholesterol, elevated triglycerides, elevated glucose (tendency for diabetes), low potassium (can cause sudden death), low magnesium (can cause sudden death), and reduction in HDL cholesterol (the "Pac-man").

For these reasons, blood pressure control should first be attempted with techniques which do not require drugs. These "non-drug" techniques should be the only therapy in people with

blood pressures above 105/70 but below 145/95.

QUESTIONS

1. What is the "safest" blood pressure?
2. What is the increase in risk if your top number is 195 compared with 105?
3. The risk of heart attack and stroke only increases if the bottom number or top number is high. T/F
4. Why do doctors only treat blood pressures that are higher than 140/90-95 with medications?
5. What are some of the harmful effects of high blood pressure medicines.

High blood pressure is almost exclusively a disease which occurs in populations who consume too much salt. See Figure 19. In populations which consume less than 1.5 gms of salt per day, high blood pressure is virtually unknown (5 gms of salt are contained in one rounded teaspoon). In societies which consume 3-4 gms of salt per day, hypertension will only develop in 3% or less. The average salt consumption in the United States is 10-15 gms per day. One fourth of the people in the United States have blood pressures above 145/95 and require medications. A far greater number of people have blood pressures in excess of the safe level of 105/70.

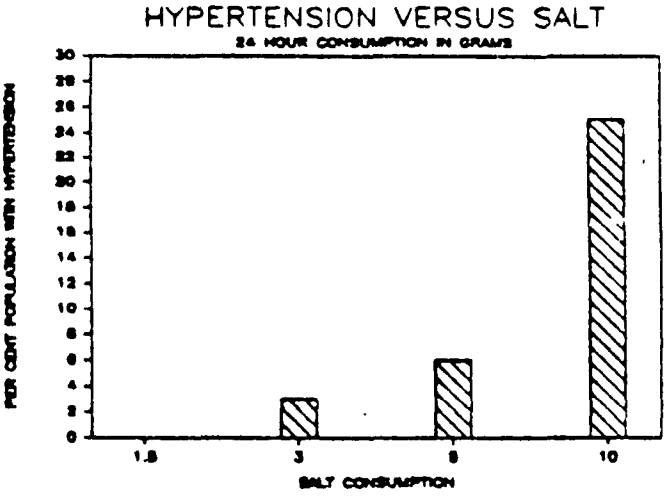


FIGURE 19

One third of the salt, which is eaten in America, is added with a salt shaker and the rest comes from restaurants and processed foods. The American Heart Association recommends that salt ingestion be limited to 4 gms per day in those without hypertension and to 2 gms per day in those with blood pressures over 145/95. Limitation of sodium to 4 gms per day can be easily accomplished by not adding salt at the table and avoiding foods, which contain high amounts of salt. Foods which contain high amounts of salt are bacon, ham, salted nuts, pop corn, potato chips, pretzels, pickles, ketchup, mustard, soy sauce, steak sauces, canned foods, processed foods, and restaurant food. MSG or mono-sodium glutamate (accent or meat tenderizer) has the same effect as salt on blood pressure.

If you have ever eaten a "low-salt" meal, you know that it tastes similar to "saw dust". Food without salt is very flat. The more salt you eat, the greater the salt content of your saliva (spit), urine, and sweat. If you are on a high salt diet, you will constantly cover your tongues with saliva (spit) which contains a great amount of salt. This high concentration of salt "deadens" the taste buds on your tongue, hence, it requires a very high salt

concentration in your food to produce the salty flavor which is so enjoyable. One to two weeks after you start a low salt diet, your tongue will be bathed with saliva (spit) which has very little salt. The sensitivity of your taste buds will increase greatly. The food will taste as good with 1/3 to 1/2 of the salt.

An analogy to illustrate this effect is the decreased sensitivity of the nose when chronically exposed to strong "smelly" odors. If you are exposed to a foul smell for a prolonged period of time, the sensitivity of your nose is lost and the foul smell is no longer appreciated (did it ever smell when you entered a bathroom but not when you left?).

Many are needlessly concerned that reduced salt intake will hamper the ability to sweat when its hot. In actuality, after the ingestion of a low salt diet, the salt content of sweat will decrease markedly so a balance will be maintained. The only exception to this are the "ultra" marathon runners, who run 50 to 60 miles in a competition. They can lose 20 quarts of sweat in a race and often require supplementation. If you sweat alot, all you need to do is to drink water. The best way to treat high blood pressure is to prevent it with a low salt diet. There are no bad "side effects" of a low salt diet. In review, the first step in a low salt diet is not to add salt to food. The second step is to avoid "salty" foods. The third step is to limit restaurant food.

QUESTIONS

1. What is your chance of developing high blood pressure if your salt ingestion is limited to 1.5 gms per day?
2. What is your chance of developing high blood pressure if your salt ingestion is limited to 4 gms pr day?
3. What is the average salt ingestion in the United States?
4. How many people in the United States have blood pressures over 140/95?
5. How many people have blood pressure over the safe pressure of 105/70?
6. How much salt is usually added at the table?
7. How much salt should you eat if you don't have high blood pressure?
8. How much salt should you eat if you have high blood pressure?
9. One teaspoon of salt weights how many grams?
10. Why does switching to a low salt diet taste so bad at first?

Another important "non-drug" therapy for control of hypertension is weight loss. Weight loss is a very effective and safe way to reduce blood pressure. Weight losses of 10-20 pounds can lower systolic and diastolic blood pressures by 30 points or more. The "side effects" of weight loss are lowered blood cholesterol, lowered blood sugar, lowered blood triglycerides, and an increase in HDL cholesterol. In essence, the side effects of weight loss are helpful for the heart and not harmful, like the side effects of many medications. The combination of weight loss and decreased salt intake is especially effective in lowering blood pressure.

Aerobic exercise can also dramatically decrease blood pressure (without a reduction in weight or salt consumption). Common aerobic exercises are walking, jogging, swimming, bike riding,

singles tennis, and aerobic dance. Football, weight lifting, baseball, and bowling are not aerobic exercises and have little effect on blood pressure or cause a slight increase. Aerobic exercise programs which benefit the cardiovascular system are performed ideally 4-5 times a week and last 20-40 minutes per session. The intensity of aerobic exercise should be such that it generates a pulse rate 2/3rds of the predicted maximal pulse rate. The predicted maximal pulse rate is equal to 205-(age/2) for those under 40 and 205-age for those over 40. For example, the exercise pulse rate for a 55 year old man should be (205-55) x 2/3 or 100. Exercise will be covered in more detail in another portion of this manual.

Reduction in alcohol consumption will lower blood pressure in many individuals. The reduction in systolic blood pressure can be as much as 25 points. Alcohol will be covered in detail in another portion of this manual. Recent evidence suggest that calcium, potassium and magnesium have important roles in hypertension. Calcium, potassium and magnesium supplementation have lowered blood pressure in certain individuals. Part of your therapy may be supplementation with one or more of these important minerals. Your doctor will give you individual advise. See Figure 20.

NON-DRUG THERAPY FOR HYPERTENSION

1. Sodium Restriction
2. Weight Loss
3. Aerobic Exercise
4. Decrease Alcohol Use
5. Calcium Supplementation
6. Magnesium Supplementation
7. Potassium Supplementation

FIGURE 20

QUESTIONS

1. What other non-drug therapy can help you lower your blood pressure?
2. What type of exercise helps lower your blood pressure?
3. What effect does exercise have on weight?
4. What effect does weight have on blood pressure?
5. If your blood pressure is 135/84 would your chances of having a heart attack be reduced, if you lowered your salt intake, lost weight, or started exercising?

In review, the risk of cardiovascular disease increases with increasing systolic and diastolic blood pressures. The risk increases with blood pressures over 105 systolic and 70 diastolic. Drug therapy will clearly benefit a person if the blood pressure is over 145/95 but may not help with blood pressures less than this. The main reason for lack of

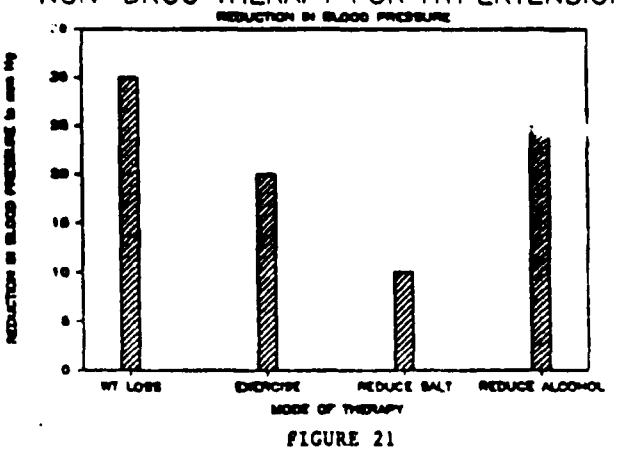


FIGURE 21

benefit in "low levels" of hypertension is due to harmful side effects of some
medications. For this reason, "non-drug" therapies, such as reduction in salt consumption, weight loss, aerobic exercise and reduction in alcohol consumption, should be used when possible in all with established hypertension and in most to prevent the development of hypertension. See Figure 21. The side effects of "non-drug" therapy for hypertension are frequently beneficial, for the heart, rather than harmful, like the side effects of several of the antihypertensive medications.

DIABETES

Diabetes is an important risk factor for the development of cardiovascular disease. The increase in risk of developing premature cardiovascular disease, if you have diabetes is 2-4 fold. Diabetes is also associated with elevated blood cholesterol, elevated triglycerides, excess weight, and low HDL cholesterol (Pac-man) levels.

It has been shown that when blood sugar rises above the normal range, there is an associated increase in serum cholesterol and triglycerides. In addition, when blood sugar rises, HDL cholesterol levels fall. When blood glucose is lowered there is a decrease in cholesterol and triglycerides as well as an increase in HDL cholesterol. Hence, controlling diabetes by not allowing the blood sugar to go too high will decrease your risk of developing premature cardiovascular disease.

There are 2 types of diabetes, juvenile onset (which requires insulin) and adult onset (does not require insulin). Eighty to eighty-five per cent of all diabetics have adult onset diabetes. The best therapy for adult onset diabetes is weight loss, avoiding sweets, low dietary fat, low dietary cholesterol, high dietary fiber, small frequent meals, and exercise. These are also helpful in those with juvenile onset diabetes, but insulin is also required. Weight loss alone, will usually control the blood sugar level of those with adult onset diabetes. The same foods which promote weight loss, are also low in fat, cholesterol and sugar.

After careful inspection, the dietary recommendations for diabetes are identical to the dietary recommendations for a cholesterol lowering diet with the additional feature of small frequent meals and greater restrictions on dietary sugar.

QUESTIONS

1. Diabetes is associated with a fold increase in risk of cardiovascular disease.
2. What are the other risk factors associated with diabetes?
3. What happens to blood cholesterol when blood sugar becomes elevated?
4. What happens to HDL cholesterol when blood sugar becomes elevated?

ASPIRIN

In up to 85% of heart attacks, blood clots cause or contribute

to the final blockage which initiates the attack Aspirin decreases the ability of the blood to clot and has been shown in many studies to decrease the risk of developing heart attacks and strokes. The reduction, in heart attacks and strokes in those who regularly take aspirin, is roughly 20-30%. The best dose of aspirin is probably 1 per day. Higher doses of aspirin do not provide any more protection against these blood clots, but clearly produce more side effects. Aspirin should not be taken by individuals who have had a bleeding ulcer, aspirin induced asthma, or in those who take the blood thinner called coumadin.

ESTROGEN

Women live 8 years longer than men on the average. Almost half of this increase is due to lower rates of cardiovascular disease. Men under age 50, have ten times as many heart attacks as women. Some of this increase in risk in men is due to higher rates of tobacco consumption and higher blood pressures.

In the late 50's birth control pills were developed with extremely high amounts of estrogen. These high amounts of estrogen were associated with an increase in the risk of developing high blood pressure, blood clots, strokes, and heart attacks, particularly in women who smoked. (The newer birth control pills use far less estrogen and other female hormones. Their use is actually safer than not using any method of contraception or pregnancy in women under age 35. In women, who smoke and are older than age 35, there is a modest increase in risk with current birth control formulations.) For these reasons, most estrogen use was condemned by the medical and lay press in the 60's and early 70's.

The Framingham Study and many other studies, showed that after a women goes through the "change of life", there is a dramatic increase in the risk of developing a heart attack. It has also been shown that women, who are not placed on low dose estrogens after the "change in life", have 3 times as many heart attacks as women who are placed on low dose hormones. Furthermore, if a woman has her ovaries removed at an early age, and is not given small physiologic doses of estrogen, there is a 5 fold increase in risk of developing a heart attack, when compared to a woman who is given estrogen replacement.

Many studies show that estrogen therapy in women, who have gone through the "change of life", lowers total cholesterol and increases HDL cholesterol (Pac man). This is a beneficial effect. The conflicting effects of estrogen may be explained as follows. High doses of estrogen are dangerous because they cause a large increase in the tendency for blood to clot, diabetes, and an increase in blood pressure. Lower doses of estrogen are helpful because they lower blood cholesterol and increase HDL cholesterol. Low dose estrogens do not increase blood pressure, increase blood sugar or increase the tendency of the blood to clot.

Testosterone, the male hormone, raises total cholesterol and lowers HDL cholesterol. It has been suggested that the different effects of testosterone and estrogen on blood cholesterol and HDL cholesterol are partly responsible for the difference in heart attack rates between men and women.

Estrogen also prevents the loss of calcium from the bones in

women who have gone through the "change of life". This keeps the bones strong and prevents fractures. Estrogen also relieves the "hot flashes" and depression associated with the "change of life". For these reasons, it is currently recommended that most women be cycled on low dose estrogens and progesterone after going through the "change of life" unless there is a reason against their use (such as breast cancer, cancer of the uterus, or history of blood clots).

QUESTIONS

1. Aspirin has been shown to decrease heart attacks and strokes. T/F
2. In % of heart attacks, a blood clot causes or contributes to the cause of the attack.
3. How does aspirin benefit the cardiovascular system?
4. What is the recommended dose of aspirin?
5. Who should not take aspirin?
6. High doses of estrogen are associated with an increase in
7. After the "change of life" there is a dramatic increase in the risk of developing a heart attack. T/F
8. Estrogen has what effect on serum HDL cholesterol?
9. Testosterone has what effect on serum HDL cholesterol?
10. What may explain part of the difference between the risk of heart attacks between men and women?

ALCOHOL

Alcohol has complex effects on the cardiovascular system. If one totally abstains from alcohol, one will be 2-3 times more likely to have a heart attack than those who drink 2 or 3 drinks per day. If one drinks 6 or more per day, one will be 3 times more likely to have a heart attack than if one totally abstaines. Overall, the lowest mortality is in those who average 1 or 2 drinks per day. See Figure 22.

Low doses of alcohol stimulate your liver and raise serum HDL levels (HDL is made in the liver). Higher doses of alcohol poison your liver, which increases total cholesterol and decreases HDL cholesterol. Furthermore, blood pressure increases with increasing consumption of alcohol. Only 20 per cent of those who totally abstain from alcohol, will have blood pressures above 145/95. Fifty per cent of those who average 6 or more drinks per day, will have blood pressures above 145/95. See Figure 23.

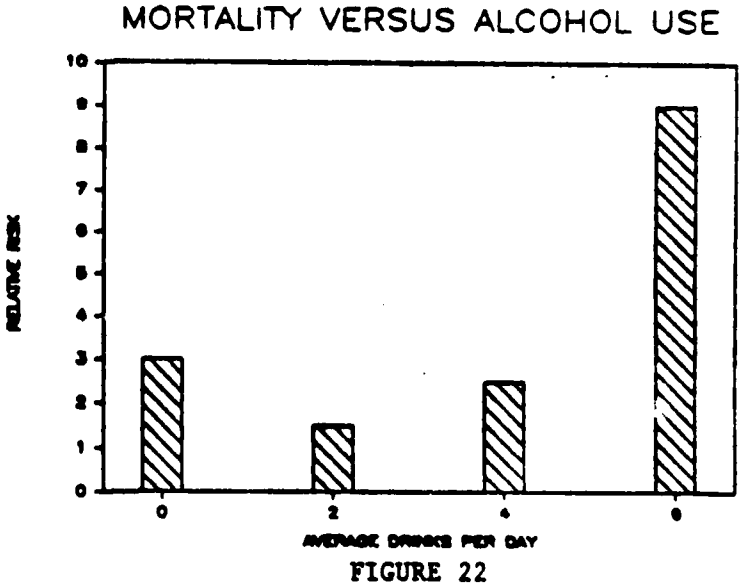


FIGURE 22

If you have high blood pressure and consume 4-6 drinks per day, your blood pressure may fall considerably if you decrease your alcohol consumption to 1 or 2 drinks per day. The reduction in blood pressure will be realized within 2 days of the reduction in alcohol consumption.

The protective effect of "low dose" alcohol is probably caused by its effect on HDL cholesterol. The harmful effect of high dose alcohol is caused by its adverse effects on blood pressure, cholesterol and HDL cholesterol.

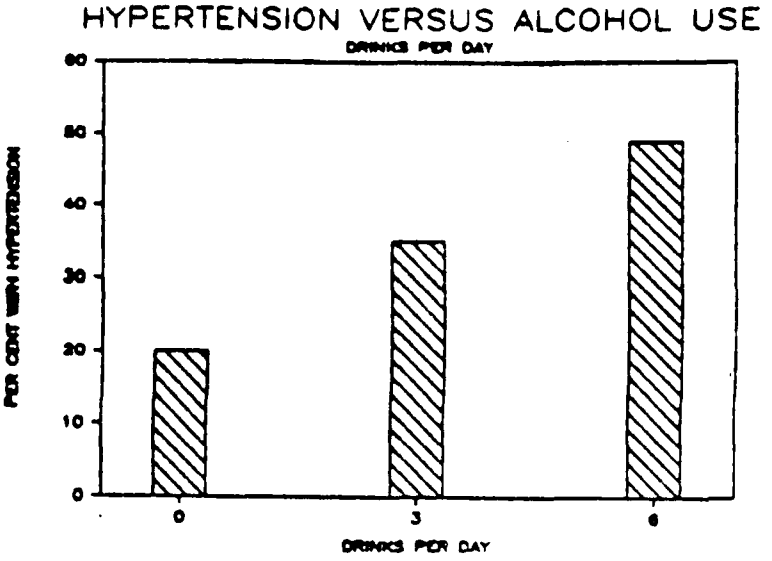


FIGURE 23

Alcohol abuse causes cirrhosis (liver failure), increases the risk of bleeding internally, and increases the risk of cancer of the mouth, throat, esophagus (food tube), liver and pancreas. It is also associated with deaths due to suicide and accidents. It is estimated that alcohol causes one half of all fatal traffic accidents in this country each year. Alcoholism is present in 10% of all males over age 40 in the United States. Certain ethnic groups have greater problems with alcohol. If you have a family member (parent or sibling) who abuses alcohol, you are at greater risk of developing alcoholism than the general population. Alcohol can be a dangerous drug if ingested by those in these high risk groups. If you do not drink and have a "risk factor" for alcoholism, than you should probably continue to avoid alcohol. If you do not have any risk factors for alcoholism, do not have hypertension, do not have any liver disease, and have low HDL cholesterol levels, you may want to consider a trial of alcohol under medical supervision. Ask your doctor about this and seek his advise.

QUESTIONS

1. Alcohol consumption can favorably affect cardiovascular risk. T/F
2. Alcohol consumption can adversely affect cardiovascular risk. T/F
3. How can alcohol consumption improve cardiovascular risk?
4. How can alcohol increase cardiovascular risk?
5. Modest consumption of alcohol is advisable for all. T/F
6. What is modest consumption of alcohol?

STRESS AND PERSONALITY

Stress and personality have been associated with the premature development of cardiovascular disease. "Type A" personality in the Framingham Study was associated with a 2 fold increase in the risk of cardiovascular disease. "Type A" personality is characterized

by competitive nature, intense striving for achievement, easily provoked hostility etc.. Individuals who have "type B" personality are relaxed, unhurried, less easily provoked and are generally more "laid back".

Several other studies have evaluated the effect of personality on the risk of cardiovascular disease but found no correlation between personality type and the development of cardiovascular disease. This subject is being actively researched.

It is clear that if you react to stress with increased cigarette consumption, excess alcohol consumption, poor dietary habits, avoidance of exercise, and weight gain, then an increase in premature cardiovascular disease can be expected. Much more research is needed in this area.

PHYSICAL ACTIVITY

Physical inactivity is a major risk factor for cardiovascular disease. In a recent study, the inactive graduates of Harvard University were 2 to 4 times more likely to develop premature cardiovascular disease, than the active graduates of Harvard. The study also showed that there was a plateau effect, i.e., leveling off, of risk reduction with exercise. That is, above a certain amount of exercise per week, there was no further reduction in cardiovascular risk.

Unfortunately, exercise will not make you immune to cardiovascular disease. The protective effect of exercise can be overwhelmed by other risk factors such as diet, smoking and high blood pressure. Jim Fix, the famous author of THE JOY OF RUNNING, died in his fifties of a heart attack. He frequently stated that he could eat all the eggs and butter he wanted because he could "run it off". Obviously he was wrong (he did live far longer than the majority of those in his family).

Exercise has many favorable effects on cardiovascular risk factors. First, exercise helps control weight. Secondly, exercise helps lower blood pressure. Thirdly, aerobic exercise increases your blood HDL cholesterol (the Pac-man). Fourthly, aerobic exercise lowers your blood sugar. Fifthly, aerobic exercise increases the efficiency of your heart and lungs, which enables them to do the same amount of work with less effort. Sixthly, exercise increases the blood supply to your heart. Seventhly, aerobic exercise can decreases your psychologic stress, make you feel good, reduce your neuromuscular tension, reduce depression and improve your body image.

There are some risks when you start a formal exercise program. The first major risk is damage to your muscles, bones, tendons and ligaments. These can be reduced by starting an exercise program slowly, and advancing the length and intensity of the exercise sessions gradually. The other major risk is heart attack and sudden death.

In general, formal exercise programs are safe with an estimated 4 deaths per every 10,000,000 exercise sessions. A recent study showed the risk of sudden death to be increased 5 fold during vigorous exercise in those who exercised regularly. There was a 56 fold increase in risk of sudden death, while vigorously exercising in those who were habitually inactive. However, those

who did not exercise regularly, were 2 1/2 times more likely to die suddenly, than those who exercised regularly. It is now clear that it is "risky" to exercise but it is more "risky" not to exercise.

The American College of Sports Medicine recommends that a stress test be performed on all inactive people over age 35, before starting a formal exercise program. Unfortunately, several people have died suddenly during vigorous exercise with a normal stress test within the previous year. Furthermore, many completely normal individuals have "abnormal" stress tests.

Recent studies from Brooks AFB and the School of Aerospace Medicine, suggest that the best proceedure is not to perform a stress tests on those who do not have symptoms suggestive of heart disease or a history of cardiovascular disease. The safest proceedure is to educate individuals on the early symptoms of heart disease, such as, chest pain, jaw pain, shoulder pain, "indigestion" etc., and start an exercise program at low exercise levels. The exercise sessions should be advanced very slowly. If any symptoms develop which suggest heart disease, than a formal evaluation should be performed by the person's doctor.

A stress test and a doctor's evaluation should be performed on all with known heart disease or symptoms suggesting heart disease, before starting an exercise program.

Not all exercise programs need to be formal. A benefit can be obtained by increasing normal daily activity. For instance, one should take the stairs rather than an elevator when possible. One should use a push mower rather than a self-propelled mower, or park the car a mile from work and walk the last mile, etc.

The exercise which should be performed is aerobic exercise. Aerobic exercise involves the use of large muscle groups for prolonged periods of time. Examples of aerobic exercise are brisk walking, running, stationary or mobile cycling, swimming, cross country skiing, rope skipping, rowing, aerobic dance etc. Baseball, football, bowling, and weight lifting are not aerobic exercises.

The exercise intensity should be great enough to increase your pulse rate to 65-80 per cent of your maximum heart rate. (Individuals, taking Inderol or other high blood pressure medications which slow the pulse rate, will not be able to use pulse rate as a guide for exercise intensity. They should exercise at an intensity which they judge to be "moderate". If you have any questions about your exercise pulse rate, see you doctor for specific advise.) Your maximum heart rate can be directly measured through stress testing or estimated from your age. The formula generally used to estimate maximum pulse is 205-(age/2) if you are under age 40 and 205-age if you are older than forty. You should exercise 20 - 40 minutes per session. The optimum amount of exercising is 5 days per week. Exercising every day of the week is no better for your heart, than exercising 5 days per week. Exercising every day of the week, produces more injuries to the muscles, bones etc. than exercising 5 days per week. Your body needs alittle time to rest and heal.

Generally, the harder you work or exercise, the shorter the duration required for heart benefit. The harder you exercise, the more likely you are to injure yourself. Furthermore, the more vigorous the exercise, the greater the risk of heart attack. For these reasons, you should not exercise harder than a level which

will increase your heart rate over 80% of the maximum (unless you are training for athletic competition).

When you start an exercise program, it is advisable to start at a heart rate which is 60% of maximum and exercise for only 10 - 15 minutes. You should exercise 3 times per week. The frequency, intensity and duration of the exercise sessions are then gradually increased over several months until you are exercising 5 times per week, for 20-40 minutes, at 65 - 80% of your maximum pulse rate. There is no further increase in benefit to your heart when exercising more frequently, harder, or for longer durations of time than this. You only increase the risk of injury.

Injuries are less likely with walking, cycling or swimming and are the preferred exercises if you have arthritis or have been inactive physically for a long period of time. Injuries can also be reduced by using specially cushioned shoes for walking or jogging.

Prolonged fatigue following a workout may indicate that your sessions are too long, too frequent, or too intense. Your sessions should be reduced in frequency, duration and/or intensity. If chest pain (if it does not change with breathing, cough, or arm movement), jaw pain, neck pain, left arm pain, left shoulder pain, "indigestion", or unusual shortness of breath occur during exercise see your physician immediately.

Exercise has many benefits. For the vast majority of people, its regular performance is safer than not exercising. There are some individuals for whom exercise is dangerous. A gradual increase in exercise and careful attention to one's body, will usually identify the person for whom exercise is dangerous.

QUESTIONS

1. Type A personality is definately associated with an increase in risk of premature cardiovascular disease. T/F
2. How can stress increase the risk of cardiovascular disease?
3. What is the increase in risk in an inactive person compared with an active person?
4. The greater the exercise the greater the benefit to the body. T/F
5. A physical education teacher recently stated, "I don't worry about what I eat, I'll just run it off." Was he correct? T/F
6. Name 5 potential ways that exercise can help your body.
7. What are the 2 main risks of an exercise program?
8. What is the increase in risk while vigorously exercising in a person who is inactive most of the time?
9. What is the increase in risk while vigorously exercising in a person who exercises regularly?
10. It is more risky to inactive than active. T/F
11. What type of exercise should be performed?
12. Who should definately receive a stress test before starting an exercise program?
13. What is the optimum frequency for aerobic exercise?
14. What is the target heart rate for aerobic exercise?
15. How can you reduce the risk of musculoskeletal injury?
16. What is the optimum duration of aerobic exercise?
17. What symptoms should make a person report to medical attention immediately?

GENETICS

Persons with parents or siblings with premature cardiovascular disease have an increase in risk of developing cardiovascular disease. If one of your parents developed cardiovascular disease before age 60, then you are at increased risk. If both of your parents developed premature cardiovascular disease, there is additional risk. In addition, if one of your parents or a sibling have a risk factor for cardiovascular disease (such as hypertension, high cholesterol, diabetes etc.), then you are at greater risk for developing this risk factor. The increase in risk comes from similar "lifestyles" as well as a genetic (inherited) tendency.

We do not know all the risk factors and all of the protective factors. There is much to be learned about preventive cardiology. If you have a strong family history of cardiovascular disease, then you should adopt a "healthy heart" lifestyle, even if no obvious "risk factors" are currently present. The healthy heart lifestyle includes low sodium, low cholesterol, low simple sugar, and low saturated fat diet. You should maintain ideal body weight and exercise regularly. Tobacco should not be used in any form. The best way to be healthy is to stay healthy. The statement "if you want a job well done, do it yourself" is particularly true in reference to your heart.

VITA

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